







22102387760











TRANSACTIONS  
OF  
THE MEDICAL SOCIETY  
OF  
LONDON.

VOLUME THE SIXTEENTH.



EDITED BY  
WILLIAM PASTEUR, M.D.  
AND  
CHARLES BARRETT LOCKWOOD, F.R.C.S.

LONDON:  
PRINTED FOR THE SOCIETY,  
BY HARRISON AND SONS, ST. MARTIN'S LANE,  
Printers in Ordinary to Her Majesty.

1893.



WELLCOME INSTITUTE LIBRARY	
Coll.	weIMOmec
Call No.	




## ADVERTISEMENT.

---

THE present volume comprises the Transactions of the Society during its one hundred and twentieth Session, from October 17th, 1892, to May 1st, 1893.





Digitized by the Internet Archive  
in 2021 with funding from  
Wellcome Library



# CONTENTS.

---

	PAGE
ADVERTISEMENT . . . . .	iii
LIST OF ILLUSTRATIONS . . . . .	x
LIST OF THE OFFICERS AND MEMBERS OF THE COUNCIL FOR SESSION 1892—1893 . . . . .	xi
LIST OF THE PRESIDENTS OF THE SOCIETY . . . . .	xiii
DECEASED BENEFACTORS OF THE SOCIETY . . . . .	xiv
LIST OF THE LETTSOMIAN LECTURERS . . . . .	xv
LIST OF THE ORATORS OF THE SOCIETY . . . . .	xviii
LIST OF THE FOTHERGILLIAN GOLD MEDALLISTS . . . . .	xx
LIST OF THE HONORARY FELLOWS OF THE SOCIETY . . . . .	xxi
LIST OF THE CORRESPONDING FELLOWS OF THE SOCIETY . . . . .	xxiii
LIST OF THE SUBSCRIBING FELLOWS OF THE SOCIETY . . . . .	xxv
LIST OF THE NON-SUBSCRIBING FELLOWS OF THE SOCIETY . . . . .	xliv
GENERAL MEETING— March 6th, 1893 . . . . .	xlvi

## COMMUNICATIONS :—

### 120TH SESSION.

1892.

#### October 17th—

- Opening Address on Names, Definitions, and Classifications.  
By the President, JONATHAN HUTCHINSON, F.R.C.S., F.R.S. . . . . 1

#### October 24th—

- A case of Volvulus of the Small Intestine, following a fall,  
successfully treated by Abdominal Section. By GEORGE R.  
TURNER, F.R.C.S. . . . . 16
- The Treatment of the Peritoneum in Abdominal Surgery. By  
W. A. MEREDITH, M.C. . . . . 21

# VI

1892.

PAGE

October 31st—

- Intra-Thoracic Auscultation : a new departure in Physical  
Diagnosis. By BENJAMIN WARD RICHARDSON, M.D., F.R.S. . 31
- The Surgical Treatment of Cysts of the Vulvo-Vaginal or  
Cowper's Glands. By ALBAN DORAN, F.R.C.S. . 38

November 7th—

- Tubal Moles and Tubal Abortions. By J. BLAND SUTTON,  
F.R.C.S. . 48

November 21st—

- Congenital Syphilis as a cause of Nervous Diseases in Children.  
By W. B. HADDEN, M.D. . 59

November 28th—

- Athletic Exercises as a cause of Disease of the Heart and Aorta.  
By WILLIAM COLLIER, M.D. . 64
- Piles : the importance of recognising the varieties as determin-  
ing the selection of Treatment. By HERBERT W. ALLINGHAM,  
F.R.C.S. . 73

December 5th—

- Cholera : its Epidemic Progression and Causation. By  
Brigade-Surgeon-Colonel J. B. HAMILTON, M.D. . 80

December 12th—

- The Irregular Heart : a Clinical Study. By A. ERNEST  
SANSOM, M.D. . 100

1893.

January 9th and 23rd, and February 6th—

- The Lettsomian Lectures : on Syphilitic Affections of the  
Nervous System. By JOHN S. BRISTOWE, M.D., F.R.S. . 116

February 13th—

- On the Prevention of Shortening and other forms of Mal-union  
after Fracture, by the use of Metal Pins passed into the Frag-  
ments subcutaneously. By C. B. KEETLEY, F.R.C.S. . 181
- The Physiology of Death from Traumatic Fever : a Study in  
Abdominal Surgery. By JOHN D. MALCOLM, C.M. . 188



## February 20th—

- On some clinical varieties of Chronic Albuminuria, chiefly with regard to Prognosis. By C. H. RALFE, M.D. . . . 212

## March 6th—

- Suprapubic Prostatectomy. By G. BUCKSTON BROWNE, M.R.C.S. . . . . 226

## March 13th—

- The Treatment of Lupus of the Face by Free Removal and Skin Grafting with Large Flaps. By W. BRUCE CLARKE, F.R.C.S. 238  
Two cases of Abdominal Section for Tumours which presented unusual characters. By W. H. BATTLE, F.R.C.S. . . . 244

## March 20th—

- Symptoms of Mental Dissolution. By GEORGE H. SAVAGE, M.D. . . . . 252  
Constitutional differences between Boys and Girls, and their relation to Educational Requirements. By FRANCIS WARNER, M.D. . . . . 263

## March 27th—

- On a case of Aphasia from a Fall on the left side of the Head. By C. E. BEEVOR, M.D. . . . . 272  
Croupous Pneumonia in Children. By FRANCIS HAWKINS, M.B. 277

## April 10th—

- Address on Neurology and Therapeutics. By W. R. GOWERS, M.D., F.R.S. . . . . 300

## April 17th—

- Pyrexia following the Anæmia due to Hæmorrhage. By M. HANDFIELD-JONES, M.D. . . . . 312  
A further communication on Hæmorrhage from Ulcerating Bubo of the Groin. By A. MARMADUKE SHEILD, F.R.C.S. . 318

## April 24th—

- On the Diagnostic Significance of Hæmoptysis in Aortic Aneurysm. By T. GILBART SMITH, M.D. . . . . 324

## May 1st—

- The Annual Oration—Physics and Letters. By W. MITCHELL BANKS, M.D., F.R.C.S. . . . . 327

# VIII

PAGE

## CLINICAL EVENINGS :—

1892.

November 14th—

Case of Tubercular Ulceration of the Pharynx successfully treated with Lactic Acid. By PERCY KIDD, M.D. . . . .	343
Two cases of Left Inguinal Colotomy. By D. H. GOODSALL, F.R.C.S. . . . .	344
“Caisson” work in Bladder Surgery. By E. HURRY FENWICK, F.R.C.S. . . . .	345
Case of Radical Cure of Hernia. By C. B. LOCKWOOD, F.R.C.S.	345
Case of Bony Tumour of the Ascending Ramus of the Ischium associated with Disease (Charcot's) of the Hip. By GEORGE R. TURNER, F.R.C.S. . . . .	346
Case of Leprosy improving under Treatment. By P. S. ABRAHAM, M.D. . . . .	348
Case of Pulsatile Tumour of the Neck. By G. A. HERSCHELL, M.D. . . . .	348
Cases by A. MARMADUKE SHEILD, F.R.C.S.—	
(a.) Cystic Tumour of the Auricle . . . . .	348
(b.) Ulceration of the edges of the Ears . . . . .	349

1893.

January 30th—

Illustrations of various Diseases. By the President, JONATHAN HUTCHINSON, F.R.S. . . . .	349
Umbilical Fistula in an Infant. By LEONARD G. GUTHRIE, M.B.	350
Case of Extreme Defect of Speech in a Boy. By WALTER B. HADDEN, M.D. . . . .	350
Cases by HERBERT W. ALLINGHAM, F.R.C.S.—	
(a.) Compound Depressed Fracture of the Skull . . . . .	351
(b.) Inguinal Colotomy . . . . .	351
Cases by CHARLES STONHAM, F.R.C.S.—	
(a.) Keloid . . . . .	351
(b.) Congenital Sacral Tumour . . . . .	351
Case of Ulceration of Auricles. By C. B. LOCKWOOD, F.R.C.S.	352
Case of Acne-Keloid (Bazin). By JAMES STARTIN, M.R.C.S. . . . .	352
Case for Diagnosis. By A. MARMADUKE SHEILD, F.R.C.S. . . . .	353
Case of Pseudo-Hypertrophic Paralysis. By W. PASTEUR, M.D.	353

February 27th—

Case of Leprosy in course of Recovery. By the President, JONATHAN HUTCHINSON, F.R.S. . . . .	354
--	-----



## IX

1893.	PAGE
Case of Acute Osteo-Myelitis of each Humeral Diaphysis ; Double Resection ; Recovery. By EDMUND OWEN, F.R.C.S. .	355
Case of Non-Malignant Stricture of the Rectum, with Pleuritic Effusion ; Thirteen Tappings ; subsequent Left Iliac Colotomy. By F. DE HAVILLAND HALL, M.D., and D. H. GOODSALL, F.R.C.S. . . . .	356
Case of Traumatic Rupture of the Urethra. By A. PEARCE GOULD, M.S. . . . .	357
Case of Chronic Enlargement of the Spleen. By EDMUND CAUTLEY, M.B. . . . .	358
Case of a Probable Syphilide refractory to Treatment. By P. S. ABRAHAM, M.D. . . . .	358
Case of Cutaneous Nævus of left Upper Extremity, with general Enlargement of the Limb . . . . .	359
Case of Dupuytren's Fracture. By JONATHAN HUTCHINSON, Jun., F.R.C.S. . . . .	359
Cases by A. MARMADUKE SHEILD, F.R.C.S.—	
(a.) Congenital Tumour in an Infant . . . . .	360
(b.) Congenital Warty Growths of the Hand and Foot.— Fungating Sore. By A. MARMADUKE SHEILD, F.R.C.S. . . . .	360
Case of Cured Meningocele. By STEPHEN PAGET, F.R.C.S. .	361
April 17th—	
Case of Excision of the Clavicle for Sarcoma. By W. F. HASLAM, F.R.C.S. . . . .	361
Sarcoma of the Clavicle. By F. BOWREMAN JESSETT, F.R.C.S.	362
April 24th—	
Case of Nasal Polypus of unusual form and size, with speci- mens and drawings. By W. SPENCER WATSON, F.R.C.S. .	363
Case after Amputation of the Hip-Joint. By H. P. SYMONDS, F.R.C.S. . . . .	366
Case of Arterio-Venous Aneurysm. By CHARLES STONHAM, F.R.C.S. . . . .	367
Cases by JONATHAN HUTCHINSON, Jun., F.R.C.S.—	
(a.) Chancre of the Cheek—Infection through a Bite .	367
(b.) Plastic Operation for Rodent Ulcer . . . . .	367

## LIST OF ILLUSTRATIONS.

---

	PAGE
Five drawings to illustrate Mr. Bland Sutton's paper on " Tubal Moles and Tubal Abortions " . . . . .	49—55
Series of pulse-tracings to illustrate Dr. Sansom's paper on " The Irregular Heart " . . . . .	113
Three drawings to illustrate Mr. Keetley's paper on " The Prevention of Shortening after Fracture " . . . . .	184, 185
Three statistical charts referring to Dr. Hawkins' paper on " Croupous Pneumonia in Children " . . . . .	281—292
Copy of photograph of patient in Mr. Sheild's case of " Con- genital Tumour in an Infant " . . . . .	360
Two drawings to illustrate Mr. Spencer Watson's case of " Nasal Polypus " . . . . .	363



OFFICERS AND COUNCIL  
OF  
THE MEDICAL SOCIETY OF LONDON,  
ELECTED MARCH, 1893.

**PRESIDENT.**

JOHN SYER BRISTOWE, M.D., F.R.S.

**VICE-PRESIDENTS.**

F. DE HAVILLAND HALL, M.D.	FREDERICK T. ROBERTS, M.D.
DAVID HENRY GOODSALL.	FREDERICK TREVES.

**HON. TREASURER.**

ARTHUR EDWARD DURHAM.

**HON. LIBRARIAN.**

WILLIAM HENRY ALLCHIN,  
M.D., F.R.S.E.

**ORATOR.**

WILLIAM MILLER ORD, M.D.

**LETTESOMIAN LECTURER.**

FREDERICK TREVES, F.R.C.S.

**COUNCIL.**

HENRY FREDERICK BAILEY.	GEORGE ALLAN HERON, M.D.
CHARLES ALFRED BALLANCE.	JONATHAN HUTCHINSON, F.R.S.
G. BUCKSTON BROWNE.	A. COOPER KEY, M.D.
JOHN CAHILL.	STEPHEN MACKENZIE, M.D.
WILLIAM COLLIER, M.D. (Oxford).	EDWARD D. MAPOTHER, M.D.
CHARLES JAMES CULLING- WORTH, M.D.	H. MONTAGUE MURRAY, M.D.
F. SWINFORD EDWARDS.	A. MARMADUKE SHEILD.
T. COLCOTT FOX, M.B.	GEORGE R. TURNER.
ARCHIBALD E. GARROD, M.D.	SAMUEL WEST, M.D.
GERALD S. HARPER, M.B.	WALTER WHITEHEAD (Man- chester).

**HONORARY SECRETARIES.**

WILLIAM PASTEUR, M.D.

CHARLES B. LOCKWOOD.

HON. SECRETARY FOR FOREIGN CORRESPONDENCE.

HEINRICH PORT, M.D.

**TRUSTEES.**

*Of the Real Estate.*

CHARLES JOHN HARE, M.D.  
THOMAS BRYANT.  
CHARLES E. BEEVOR, M.D.

*Of the Personal Property.*

CHARLES H. F. ROUTH, M.D.  
T. GILBART SMITH, M.D.  
EDMUND OWEN.

CHAIRMAN OF HOUSE AND FINANCE COMMITTEE.

DAVID HENRY GOODSALL.

THE ABOVE CONSTITUTE THE COUNCIL.

**REGISTRAR.**

WILLIAM R. HALL.

**LIBRARY COMMITTEE.**

WILLIAM HENRY ALLCHIN, M.D., F.R.S.E. (*Hon. Librarian*), CHAIRMAN.  
 CHARLES E BEEVOR, M.D.                      WILLIAM ADAMS FROST.  
 AMAND ROUTH, M.D.                              J. BLAND SUTTON.

**PUBLICATION COMMITTEE.**

W. H. BATTLE.                                      F. G. D. DREWITT, M.D.  
 CLINTON T. DENT.                                SIR W. ROBERTS, M.D., F.R.S.

**HOUSE AND FINANCE COMMITTEE.**

DAVID HENRY GOODSALL, CHAIRMAN.

THE PRESIDENT.

THE TRUSTEES OF THE PERSONAL PROPERTY.

THE TREASURER.

CHARLES A. BALLANCE.                      A. M. SHEILD.  
 THOMAS BRYANT.                              SAMUEL WEST, M.D.

**COMMITTEE OF REFEREES.**

MEDICINE.

J. MITCHELL BRUCE, M.D.                      SIDNEY COUPLAND, M.D.  
 T. LAUDER BRUNTON, M.D.,                  J. KINGSTON FOWLER, M.D.  
 F.R.S.    CHARLES H. RALFE, M.D.

SURGERY.

CHARLES A. BALLANCE.                      FREDERICK TREVES.  
 W. MITCHELL BANKS.                          W. J. WALSHAM.  
 A. M. SHEILD.

MIDWIFERY.

C. J. CULLINGWORTH, M.D.                  AMAND ROUTH, M.D.  
 W. DUNCAN, M.D.                              JOHN WILLIAMS, M.D.  
 A. H. N. LEWERS, M.D.

N.B.—The Honorary Secretaries are *ex officio* Members of all Committees.



THE PRESIDENTS OF THE SOCIETY.

---

1773. JOHN MILLAR, M.D.  
1775. JOHN COAKLEY LETTSOM, M.D., F.R.S.  
1776. NATHANIEL HULME, M.D., F.R.S.  
1779. GEORGE EDWARDS, M.D.  
1780. SAMUEL FOART SIMMONS, M.D., F.R.S.  
1783. JOHN SIMS, M.D.  
1784. JOHN WHITEHEAD, M.D.  
1785. JOHN RELPH, M.D.  
1786. JAMES SIMS, M.D.\*  
1809. JOHN COAKLEY LETTSOM, M.D., F.R.S.  
1811. GEORGE PINCKARD, M.D.  
1813. JOHN COAKLEY LETTSOM, M.D., F.R.S.  
1815. JOSEPH ADAMS, M.D.  
1817. THOMAS WALSHMAN, M.D.  
1819. HENRY CLUTTERBUCK, M.D.  
1821. DAVID UWINS, M.D.  
1823. WILLIAM SHEARMAN, M.D.  
1825. HENRY CLUTTERBUCK, M.D.  
1827. JOHN HASLAM, M.D.  
1829. THOMAS CALLAWAY.  
1831. JOHN BURNE, M.D.  
1833. WILLIAM KINGDOM.  
1835. JOHN WHITING, M.D.  
1837. THOMAS EGERTON BRYANT.  
1839. LEONARD STEWART, M.D.  
1840. HENRY CLUTTERBUCK, M.D.  
1842. GEORGE PILCHER.  
1844. THEOPHILUS THOMPSON, M.D.  
1846. WALTER COOPER DENDY.  
1848. HENRY HANCOCK.  
1850. JAMES RISDON BENNETT, M.D.  
1851. EDWARD WILLIAM MURPHY, M.D.  
1852. JOHN BISHOP, F.R.S.  
1853. FORBES WINSLOW, M.D., D.C.L.  
1854. EDWARD HEADLAND.  
1855. JOHN SNOW, M.D.  
1856. WILLIAM DINGLE CHOWNE, M.D.  
1857. FRANCIS HIRD.  
1858. WILLIAM HUGHES WILLSHIRE, M.D.  
1859. JOHN HILTON, F.R.S.

\* *Dr. James Sims was President for twenty-two years.*

**THE PRESIDENTS OF THE SOCIETY**—*continued.*

1860. ALFRED BARING GARROD, M.D., F.R.S.  
 1861. WILLIAM COULSON.  
 1862. FRANCIS SIBSON, M.D., F.R.S.  
 1863. EDWIN CANTON.  
 1864. ROBERT GREENHALGH, M.D.  
 1865. ISAAC BAKER BROWN.  
 1866. CHARLES JOHN HARE, M.D.  
 1867. HENRY SMITH.  
 1868. BENJAMIN WARD RICHARDSON, M.D., F.R.S.  
 1869. PETER MARSHALL.  
 1870. JOHN GAY.  
 1871. ANDREW CLARK, M.D.  
 1872. THOMAS BRYANT.  
 1873. SAMUEL OSBORNE HABERSHON, M.D.  
 1874. VICTOR DE MÉRIC.  
 1875. CHARLES H. F. ROUTH, M.D.  
 1876. WILLIAM ADAMS.  
 1877. GEORGE BUCHANAN, M.D.  
 1878. ERASMUS WILSON, F.R.S.  
 1879. JOHN COCKLE, M.D.  
 1880. FREDERICK JAMES GANT.  
 1881. WILLIAM HENRY BROADBENT, M.D.  
 1882. FRANCIS MASON.  
 1883. SIR JOSEPH FAYRER, K.C.S.I., M.D., F.R.S.  
 1884. ARTHUR EDWARD DURHAM.  
 1885. WILLIAM M. ORD, M.D.  
 1886. ROBERT BRUDENELL CARTER.  
 1887. J. HUGHLINGS JACKSON, M.D., F.R.S.  
 1888. SIR WILLIAM MACCORMAC.  
 1889. CHARLES THEODORE WILLIAMS, M.D.  
 1890. JOHN KNOWSLEY THORNTON.  
 1891. RICHARD DOUGLAS POWELL, M.D.  
 1892. JONATHAN HUTCHINSON, F.R.S.  
 1893. JOHN SYER BRISTOWE, M.D., F.R.S.

**DECEASED BENEFACTORS OF THE SOCIETY.**

1778. JOHN COAKLEY LETTSOM, M.D., F.R.S., A FREEHOLD  
       HOUSE, No. 3, BOLT COURT, FLEET STREET, of the value  
       of     ....     ....     ....     ....     ....     ....     ....     ....     £2500  
 1780. ANTHONY FOTHERGILL, M.D., F.R.S.     ....     ....     £500  
 1807. NATHANIEL HULME, M.D., F.R.S.     ....     ....     £50  
 1887. PEDRO FRANCISCO DE COSTA ALVARENGA, M.D.     £500



## THE LETTSOMIAN LECTURERS.

THE LETTSOMIAN LECTURESHIP WAS ESTABLISHED IN 1850.

---

- 1851. GEORGE OWEN REES, M.D., F.R.S., On some of the Pathological Conditions of the Urine.
- „ GEORGE JAMES GUTHRIE, F.R.S., On some of the more Important Points of Surgery.
- 1852. FORBES WINSLOW, M.D., On Medico-legal Evidence in Cases of Insanity.
- „ HENRY HANCOCK, On the Anatomy and Physiology of the Male Urethra, and on the Pathology of Stricture of that Canal.
- 1854. EDWARD WILLIAM MURPHY, M.D., On Parturition as Illustrating the Importance of a Competent Education in the Practice of Midwifery.
- 1855. THEOPHILUS THOMPSON, M.D., On Pulmonary Consumption.
- „ JOHN BISHOP, F.R.S., On the Physical Constitution, Diseases, and Fractures of Bones.
- „ FRANCIS SIBSON, M.D., F.R.S., On the Influence of the Nervous System on Respiration and Circulation.
- „ FRANCIS HIRD, On some Special Points in the Anatomy of the Uterus, and its Structural Lesions the result of Inflammation.
- 1857. ALFRED BARING GARROD, M.D., F.R.S., On Illustrations of the Pathology and Treatment of Gout.
- 1858. ROBERT BARNES, M.D., On the Physiology and Treatment of Flooding from Unnatural Position of the Placenta.
- „ EDWIN LANKESTER, M.D., F.R.S., On the History, Symptoms, and Treatment of Intestinal and other Worms Parasitic on the Human Body.
- 1859. FREDERICK WILLIAM HEADLAND, M.D., On the Advance during Modern Times of the Science of Medical Treatment.
- „ VICTOR DE MÉRIC, On Syphilis.
- 1860. FREDERICK WILLIAM PAVY, M.D., F.R.S., On Certain Points connected with Diabetes.
- „ ANDW. CLARK, M.D., On Certain Evidences of the Arrestment of Phthisis.
- 1861. CHARLES JOHN HARE, M.D., Practical Observations on some of the Points of Difficulty in the Investigation of Tumours and Intumescence of the Abdomen.
- „ HENRY HAYNES WALTON, On the Application of the Ophthalmoscope, and its Advantages.
- 1862. BENJAMIN WARD RICHARDSON, M.D., F.R.S., On Certain of the Phenomena of Life.

1862. FREDERICK WILLIAM MACKENZIE, M.D., On the Pathology and Treatment of Phlegmasia Dolens.
1863. HENRY THOMPSON, On Practical Lithotomy and Lithotrity.
- „ JAMES BIRD, M.D., On Public and Private Hygiene.
1864. THOMAS BRYANT, On the Surgical Diseases of Children.
- „ CHARLES HENRY FELIX ROUTH, M.D., On some Points connected with the Pathology, Differential Diagnosis, and Treatment of Fibrous Tumours of the Uterus.
1865. HENRY SMITH, On the Surgery of the Rectum.
- „ JOHN LOUIS WILLIAM THUDICHUM, M.D., On Medicine: the Progress of Urology, with Practical Illustrations of its Value in the Diagnosis and Treatment of several Diseases.
1866. FRANCIS EDMUND ANSTIE, M.D., On certain Painful Affections of the Fifth Nerve.
1867. JOHN GAY, On Varicose Diseases and Ulcers of the Lower Extremities.
1868. GEORGE BUCHANAN, M.D., On the Diagnosis and Management of Lung Diseases in Children.
1869. WILLIAM ADAMS, On Rheumatic and Strumous Diseases of the Joints, and the Treatment for the Restoration of Motion in Partial Ankylosis.
1870. WILLIAM TILBURY FOX, M.D., On Eczema: its Nature and Treatment.
1871. FREDERICK JAMES GANT, On Excisional Surgery of the Joints; the Conditions appropriate for Excision; the Operations; After-Treatment and Results.
1872. SAMUEL OSBORNE HABERSHON, M.D., On the Pathology and Treatment of some Diseases of the Liver.
1873. HENRY LEE, On Urethral Discharges.
1874. WILLIAM HENRY BROADBENT, M.D., On Syphilitic Affections of the Nervous System.
1875. CHARLES FREDERICK MAUNDER, On the Surgery of the Arteries.
1876. CHARLES THEODORE WILLIAMS, M.D., The Influence of Climate in the Treatment of Pulmonary Consumption.
1877. ALFRED WILTSHIRE, M.D., On Vascular Rhythm as exemplified in Periodical Hæmorrhages, General and Local; and on the Treatment of Hæmorrhages from the Female Generative Organs.
1878. FRANCIS MASON, On the Surgery of the Face.
1879. JOHN CHARLES THOROWGOOD, M.D., On Bronchial Asthma: its Causes, Pathology, and Treatment.
1880. WILLIAM FREDERICK TEEVAN, On the Treatment of Stricture of the Urethra, Enlarged Prostate, and Stone in the Bladder, with special reference to Recent Progress.
1881. Sir JOSEPH FAYRER, K.C.S.I., M.D., F.R.S., On Tropical Dysentery and Diarrhœa.
1882. HUTCHINSON ROYES BELL, On Diseases of the Testicles and their Coverings.
1883. ARTHUR ERNEST SANSOM, M.D., On the Treatment of Certain Forms of Valvular Disease of the Heart.



## XVII

- 1884. ROBERT BRUDENELL CARTER, On Modern Operations for Cataract.
- 1885. T. LAUDER BRUNTON, M.D., F.R.S., On Digestive Disorders: their Consequences and their Treatment.
- 1886. JONATHAN HUTCHINSON, F.R.S., On some Moot Points in the Natural History of Syphilis.
- 1887. JOHN LANGDON-DOWN, M.D., On some of the Mental Affections of Childhood and Youth.
- 1888. REGINALD HARRISON, On some Points in the Surgery of the Urinary Organs.
- 1889. WILLIAM RICHARD GOWERS, M.D., F.R.S., On Syphilis and the Nervous System.
- 1890. EDMUND OWEN, On Selected Subjects in the Surgery of Infancy and Childhood.
- 1891. STEPHEN MACKENZIE, M.D., On Anæmia: its Pathology, Symptoms, and Treatment.
- 1892. WILLIAM ROSE, On the Surgical Treatment of Trigeminal Neuralgia.
- 1893. JOHN SYER BRISTOWE, M.D., F.R.S., On Syphilitic Affections of the Nervous System.

## THE ORATORS.

- |   |  |
|---|--|
| 1774. JOHN SIMS, M.D.                       | 1818. DAVID UWINS, M.D.                      |
| 1776. DAVID MILLAR, M.D.                    | 1819. THOMAS J. PETTIGREW, F.R.S.            |
| 1777. NATH. HULME, M.D., F.R.S.             | 1820. THOMAS HANCOCK, M.D.                   |
| 1778. JOHN COAKLEY LETTSOM,<br>M.D., F.R.S. | 1821. THOMAS CALLAWAY.                       |
| 1779. GEORGE EDWARDS, M.D.                  | 1822. JAMES COPLAND, M.D.                    |
| 1780. JOHN KOOYSTRA, M.D.                   | 1823. EDWARD GRAINGER.                       |
| 1781. SAMUEL FOART SIMMONS,<br>M.D., F.R.S. | 1824. GORDON SMITH, M.D.                     |
| 1782. LOFTUS WOOD, M.D.                     | 1825. EUSEBIUS ARTHUR LLOYD.                 |
| 1783. JOHN SIMS, M.D.                       | 1826. JOHN HASLAM, M.D.                      |
| 1784. JOHN WHITEHEAD, M.D.                  | 1827. WILLIAM KINGDOM.                       |
| 1785. JOHN RELPH, M.D.                      | 1828. JOHN BURNE, M.D.                       |
| 1787. JOSEPH HOOPER.                        | 1829. WILLIAM GREVILLE JONES.                |
| 1788. JOHN MEYER, M.D.                      | 1830. LEONARD STEWART, M.D.                  |
| 1789. RICHARD DENNISON, M.D.                | 1831. MONTAGUE GOSSETT.                      |
| 1790. GEORGE WALLIS, M.D.                   | 1832. JOHN WHITING, M.D.                     |
| 1791. SAMUEL SUTTON, M.D.                   | 1833. FREDERICK SALMON.                      |
| 1792. EDWARD FRYER, M.D.                    | 1834. WILLIAM SHEARMAN, M.D.                 |
| 1793. JAMES JAMESON, M.D.                   | 1835. WALTER COOPER DENDY.                   |
| 1794. GILBERT THOMPSON, M.D.                | 1836. WILLIAM F. BLICKE, M.D.                |
| 1795. JOHN ABERNETHY.                       | 1837. EDWARD HEADLAND.                       |
| 1796. JOHN COAKLEY LETTSOM,<br>M.D., F.R.S. | 1838. THEOPHILUS THOMPSON, M.D.,<br>F.R.S.   |
| 1797. JAMES WARE.                           | 1839. GEORGE PILCHER.                        |
| 1798. SAMUEL FERRIS, M.D., F.R.S.           | 1840. JAMES RISDON BENNETT, M.D.             |
| 1799. EDWARD FORD.                          | 1841. WM. DINGLE CHOWNE, M.D.                |
| 1800. THOMAS BRADLEY, M.D.                  | 1842. HENRY HANCOCK.                         |
| 1801. WILLIAM CHAMBERLAINE.                 | 1843. LEONARD STEWART, M.D.                  |
| 1802. JOHN SIMS, M.D.                       | 1844. THOMAS BELL, F.R.S.                    |
| 1803. JOHN ANDRÉE.                          | 1845. MARSHALL HALL, M.D.                    |
| 1804. JOHN COAKLEY LETTSOM,<br>M.D., F.R.S. | 1846. JOHN BISHOP, F.R.S.                    |
| 1805. GEORGE PINCKHARD, M.D.                | 1847. GOLDING BIRD, M.D., F.R.S.             |
| 1806. HENRY FIELD.                          | 1848. FRANCIS HIRD.                          |
| 1807. JOSEPH ADAMS, M.D.                    | 1849. WILLIAM HUGHES WILL-<br>SHIRE, M.D.    |
| 1808. JOHN MASON GOOD, F.R.S.               | 1850. FRANCIS HIRD.                          |
| 1809. SAYER WALKER, M.D.                    | 1851. RICHARD ROWLAND.                       |
| 1810. GEORGE BIRKBECK, M.D.                 | 1852. EDWIN CANTON.                          |
| 1811. WILLIAM BLAIR.                        | 1853. JOHN SNOW, M.D.                        |
| 1812. RICHARD TEMPLE, M.D.                  | 1854. HENRY SMITH.                           |
| 1813. RICHARD SAUMAREZ, F.R.S.              | 1855. JAMES FERNANDEZ CLARKE.                |
| 1814. GEORGE REES, M.D.                     | 1856. BENJ. WARD RICHARDSON,<br>M.D., F.R.S. |
| 1815. JOHN TAUNTON.                         | 1857. WILLIAM ADAMS.                         |
| 1816. HENRY CLUTTERBUCK, M.D.               | 1858. ALFRED BARING GARROD, M.D.             |
| 1817. JAMES STEVENSON.                      | 1859. CHARLES HENRY FELIX<br>ROUTH, M.D.     |



- |   |  |
|---|--|
| 1860. JOHN GAY.                               | 1879. WALTER JOHN COULSON.                         |
| 1861. ARTHUR LEARED, M.D.                     | 1880. WILLIAM HENRY BROADBENT,<br>M.D.             |
| 1862. VICTOR DE MÉRIC.                        | 1881. ARTHUR EDWARD DURHAM.                        |
| 1863. SAMUEL OSBORNE HABERSHON,<br>M.D.       | 1882. EDMUND SYMES THOMPSON,<br>M.D.               |
| 1864. JOHN LOUIS WILLIAM THUDICHUM, M.D.      | 1883. EDWARD LUND.                                 |
| 1865. ROBERT GREENHALGH, M.D.                 | 1884. CHARLES THEODORE WILLIAMS,<br>M.D.           |
| 1866. THOMAS CHRISTOPHER WEEDEN COOKE.        | 1885. GEORGE MURRAY HUMPHRY,<br>M.D., F.R.S.       |
| 1867. FREDERICK WILLIAM HEADLAND, M.D.        | 1886. RICHARD DOUGLAS POWELL,<br>M.D.              |
| 1868. WILLIAM FREDERICK TEEVAN.               | 1887. Sir WILLIAM MacCORMAC,<br>F.R.C.S.           |
| 1869. GEORGE DUNCAN GIBB, M.D.                | 1888. Sir JOSEPH FAYRER, K.C.S.I.,<br>M.D., F.R.S. |
| 1870. FRANCIS MASON.                          | 1889. JONATHAN HUTCHINSON, F.R.S.                  |
| 1871. WILLIAM CHOLMELEY, M.D.                 | 1890. ARTHUR ERNEST SANSOM, M.D.                   |
| 1872. FREDERICK JAMES GANT.                   | 1891. Sir JOSEPH LISTER, Bart.,<br>F.R.S.          |
| 1873. JOHN COCKLE, M.D.                       | 1892. Sir JAMES CRICHTON BROWNE,<br>M.D., F.R.S.   |
| 1874. ROBERT BRUDENELL CARTER.                | 1893. W. MITCHELL BANKS, M.D.                      |
| 1875. GEORGE BUCHANAN, M.D.                   |  |
| 1876. ERASMUS WILSON, F.R.S.                  |  |
| 1877. JOHN HUGHLINGS JACKSON,<br>M.D., F.R.S. |  |
| 1878. ALFRED CARPENTER, M.D.                  |  |

# THE FOTHERGILLIAN GOLD MEDALLISTS.

- |                                       |   |
|---------------------------------------|---|
| 1787. WILLIAM FALCONER, M.D.          | 1852. FREDERICK WILLIAM HEAD-<br>LAND.        |
| 1790. ROBERT WILLAN, M.D.             | 1853. ALFRED WILLIAM POLAND.                  |
| 1791. JOHN COAKLEY LETTSOM,<br>M.D.   | 1854. BENJAMIN WARD RICHARDSON,<br>M.D.       |
| 1795. JOHN MASON GOOD.                | 1856. WILLIAM BURKE RYAN.                     |
| 1801. FRANCIS BOUTTATZ, M.D.          | 1857. EDWIN CANTON.                           |
| 1803. EDWARD JENNER, M.D.             | 1858. THOMAS HERBERT BARKER,<br>M.D.          |
| 1824. ROBERT W. BAMPFIELD.            | 1859. ALDERMAN THOMAS HOUGHTON<br>WATERS.     |
| 1828. JOHN GEORGE PARRY.              | 1868. JOHN CLAY.                              |
| 1831. WILLIAM AUGUSTUS GUY.           | 1870. THOS. SMITH CLOUSTON, M.D.              |
| 1834. WILLIAM JAMES CLEMENT.          | 1872. EDWARDS CRISP, M.D.                     |
| 1835. GEORGE MOORE.                   | 1873. JOHN KENT SPENDER, M.D.                 |
| 1836. THOMAS EGERTON BRYANT.          | 1877. PETER MURRAY BRAIDWOOD,<br>M.D.         |
| 1838. GEORGE PILCHER.                 | 1878. JOHN MILNER FOTHERGILL,<br>M.D.         |
| 1840. SAMUEL OSBORN.                  | 1882. THOMAS MICHAEL DOLAN, M.D.              |
| 1842. JAMES RISDON BENNETT, M.D.      | 1883. NORMAN PORRITT.                         |
| 1843. JOHN WEAVER LEVER, M.D.         | 1886. JOHN STRAHAN.                           |
| 1844. HENRY PRATT ROBARTS.            | 1888. HOBART AMORY HARE, M.D.,<br>U.S.A.      |
| 1845. WALTER COOPER DENDY.            | 1893. WILLIAM RICHARD GOWERS,<br>M.D., F.R.S. |
| 1846. ROBERT MORTIMER GLOVER,<br>M.D. |   |
| 1847. SILAS STEDMAN.                  |   |
| 1849. JOHN MILLIGAN.                  |   |
| 1850. RICHARD PAYNE COTTON, M.D.      |   |
| 1851. RICHARD HODGES.                 |   |



## THE HONORARY FELLOWS.

- 
1893. BARNES, ROBERT, M.D., Lingwood, Liss, Hants, LL, C.
1876. BARNES, J. K., M.D., Surgeon-General U.S. Army, Washington.
1881. BILLINGS, JOHN S., M.D., Washington, Surgeon to the United States Army ; Librarian to the Surgeon-General's Library, Washington.
1881. BILLROTH, THEODORE, M.D., Professor of Surgery in the University of Vienna.
1873. CHAUVEAU, A., Professor of Physiology at the Medical School of Lyons.
1890. CRUDELI, TOMMASI, M.D., Rome.
1881. DA COSTA, J. M., M.D., Professor of Medicine in the Jeffreson Medical College, 1700, Walnut-street, Philadelphia.
1881. EMMET, THOMAS ADDIS, M.D., 89, Madison-avenue, Surgeon to the Woman's Hospital of the State of New York.
1886. GAIRDNER, WILLIAM TENNANT, M.D., LL.D. Edin., F.R.C.P. Edin., F.R.S., 225, St. Vincent-street, Glasgow.
1881. HALLA, JOSEPH, Professor of Medicine in the University of Prague.
1869. HARE, CHARLES JOHN, M.D., Berkeley House, Manchester-square, W., Consulting Physician to University College Hospital, and late Professor of Clinical Medicine in University College. P, VP 2. C 8, LL. *Trustee.*
1873. HELMHOLTZ, HERMANN LUDWIG FERDINAND, M.D., Professor of Physics and Physiological Optics in the University of Berlin.
1890. HOLMGREN, FRITHIOF, Professor, Upsala.
1883. HUMPHRY, Sir GEORGE MURRAY, M.D., F.R.S., Professor of Surgery in the University of Cambridge. O, C 2.
1873. HUXLEY, The Right Honourable THOMAS HENRY, LL.D., F.R.S., Hodeslea, Eastbourne, Professor of Biology in the Normal School of Science and in the Royal School of Mines.
1875. JENNER, Sir WILLIAM, Bart., K.C.B., D.C.L., LL.D., M.D., F.R.S., Greenwood, Durley, Hants, Physician-in-Ordinary to H.M. the Queen and to H.R.H. the Prince of Wales ; late President of the Royal College of Physicians ; Emeritus Professor of Clinical Medicine in University College, London ; Consulting Physician to University College Hospital.
1890. KOCHER, THEODOR, Professor, Berne.
1884. LARREY, Baron, M.D., Paris, Rue de Lille, 91.
1883. LE ROY DE MERICOURT, A., M.D., Paris.

- 1890. LOMBARD, HENRI-CLERMOND, M.D., Geneva.
- 1878. MITCHELL, S. WEIR, M.D., Walnut-street, Philadelphia.
- 1881. NUSSBAUM, JOHN NEPOMUK RITTER VON, M.D., Professor of Surgery in the University of Munich.
- 1875. OLLIER, Professor, Lyons.
- 1873. PAGET, Sir JAMES, Bart., D.C.L., LL.D., F.R.S., 1, Harewood-place, Hanover-square, W., Serjeant-Surgeon to H.M. the Queen ; Surgeon to H.R.H. the Prince of Wales ; Consulting Surgeon to St. Bartholomew's Hospital.
- 1876. PANCOAST, JOSEPH, M.D., 1030, Chesnut-street, Philadelphia, Professor of Anatomy in the Jeffreson Medical College.
- 1890. PASTEUR, LOUIS, Member of the Institute of France, Paris.
- 1877. SANNÉ, A., 12, Place de Laborde, Paris.
- 1881. TARNIER, STEPHANIE, M.D., Professor of Obstetric Medicine in the School of Medicine, Paris.
- 1873. TYNDALL, JOHN, F.R.S.
- 1881. VERNEUIL, AUGUSTE ARISTIDE, M.D., Professor of Medicine in the School of Medicine, Paris.
- 1873. VIRCHOW, RUDOLPH, M.D., Professor of Pathological Anatomy in the University of Berlin.

## CORRESPONDING FELLOWS.

- 
1851. ALBARO, J. MENDEZ, Madrid.  
 1882. BADALONI, GIUSEPPE, M.D., Fano, Prov. di Pesaro, Italy.  
 1856. BAKER, ALBERT, M.D., The Laurels, Pinhoe, Exeter.  
 1855. BEARDSLEY, AMOS, Bay Villa, Grange, Lancashire.  
 1850. BENAVENTE, MARIANO, Madrid.  
 BENEKE, F. W., M.D., New York.  
 1850. BÖHM, PROFESSOR, M.D., Vienna.  
 BOTTINI, GIUSEPPE, M.D., Milan.  
 1837. BUHRING, J. J., M.D., Berlin.  
 CADE, THOMAS CHARLES, Spondon, Derby.  
 1855. COATES, CHARLES, M.D., F.R.C.P., 10, Circus, Bath, Consulting Physician  
 to the Bath Royal United Hospital. c 3.  
 1850. COX, WILLIAM ISIDORE, Hawkesbury, Upton, Gloucestershires. c.  
 1876. DE MUYNCK, J., M.D., Ghent.  
 1836. ECSTEIN, SIGISMUND, M.D., Vienna.  
 EYLANDT, JOHANN EMIL, M.D., Curland, Russia.  
 1853. FALLOT, R., M.D., St. Laurent d'Aigonz, Montpellier, France.  
 1889. FRANK, PHILIP, M.D., F.R.C.P., Cannes, France.  
 1876. GRIFFITH, RICHARD GLYN, Allahabad, India.  
 1864. HASENFELD, EMMANUEL, M.D., Pesth.  
 HYMAN, —, M.D., Antwerp.  
 1851. IZGUIERDO, SEBASTIAN OBTEGA, Madrid.  
 1875. JONES, PHILIP SYDNEY, M.D., F.R.C.S., Examiner in Medicine in the  
 University of Sydney, Australia, Hon. Consulting Surgeon to the  
 Sydney Infirmary.  
 1861. JOURNEZ, HENRI, M.D., 43, Rue de la Charité, Bruxelles, Belgique.  
 1851. KÖLLIKER, ALBERT, M.D., Professor of Anatomy and Physiology at the  
 University of Wurzburg.  
 1876. LEIGHTON, WALTER H., M.D., Lowell, Massachusetts, U.S.  
 LEON, JOSE, Madrid.  
 1851. LLANOS, ANTONIO CAMPO, Madrid.  
 1851. LOVERA, JOSE, Madrid.  
 1851. MARINO, BONIFACIO MATREOS, Madrid.  
 MENDEZ, BARTHOLOME, Madrid.  
 MOLINA, M. M., Madrid.



1851. NEGRI, GAETANO, M.D., Pisa.  
ORTEGA, J. R., Madrid.
1865. PERUZZI, DOMENICO, M.D., 22, Via Mazzini, Bologna.
1882. RESTREPO, ALESSANDO EDUARDO, M.D., Medellin, Columbia, U.S.A.
1886. ROCHA, A., M.D., Coimbra, Beira, Portugal.
1860. ROUSSEL, M.D., Dean of the Faculty of Medicine, Montpellier.  
SCHARLAN, GUS. W., M.D., Stettin, Prussia.
1876. SCHMITZ, RICHARD, M.D., Neuenahr.
1874. SCHUTGOWSKY, J., St. Petersburg.
1851. SESSE, M., Mesqui, Madrid.  
STOCKWELL, THOMAS GOLDESBROUGH, F.R.C.S., 6, Circus, Bath,  
Surgeon to the Bath Royal United Hospital.
- TEREZA, FELIX GARCIA, Madrid.
- VALDEZ, FRANCO CORTIGO, Madrid.
- WILLIAMS, CHARLES, F.R.C.S. Edin., 48, Prince of Wales-road,  
Norwich; Surgeon to the Norfolk and Norwich Hospital.

# THE FELLOWS

## OF

### THE MEDICAL SOCIETY OF LONDON.

---

#### EXPLANATION OF ABBREVIATIONS.

P.—PRESIDENT.	FM.—FOTHERGILLIAN GOLD MEDALLIST
VP.—VICE-PRESIDENT.	SM.—SILVER MEDALLIST.
T.—TREASURER.	O.—ORATOR.
L.—LIBRARIAN.	CFC.—CHAIRMAN, HOUSE AND FINANCE
S.—SECRETARY.	COMMITTEE.
C.—COUNCILLOR.	§—SEC. FOR FOREIGN CORRESPONDENCE.
LL.—LETTSOMIAN LECTURER.	*—LIFE MEMBERS.
	TR.—TRUSTEE.

The number prefixed signifies the date of election. The figures appended indicate the number of Sessions served, and refer to past appointments ONLY.

---

1890. ABBOT-ANDERSON, WILLIAM MAURICE, M.B., 10, Old Cavendish-street, Cavendish-square, W.
1888. ABBOTT, CHARLES EDWARD, M.R.C.S., Shrapnels, Taunton.
1891. ABRAHAM, PHINEAS S., M.D., 2, Henrietta-street, Cavendish-square, W.
1890. ACKLAND, ROBERT CRAIG, M.R.C.S., 13, Savile-row, W.
1883. ACLAND, THEODORE DYKE, M.D., 74, Brook-street, Grosvenor-square, W. c.
1884. ADAM, JAMES, M.D., Mallings-place, West Mallings, Kent.
1889. ADAMS, JAMES, M.D., 4, Chiswick-place, Eastbourne.
1878. ADAMS, JOSIAH OAKE, M.D., Brook House, Upper Clapton, E.
1852. \*ADAMS, WILLIAM, F.R.C.S., 5, Henrietta-street, Cavendish-square, W. p, c 8, o, vp 3, LL.
1878. \*ALLCHIN, WILLIAM HENRY, M.D., F.R.S.E., 5, Chandos-street, Cavendish-square, W. vp 2, *Hon. Librarian*.
1873. ALLEN, HENRY MARCUS, F.R.C.P. Edin., 20, Regency-square, Brighton.
1873. ALLFREY, CHARLES HENRY, M.D., Plas Newydd, St. Leonards-on-Sea.
1883. ALLINGHAM, HERBERT W., F.R.C.S., 25, Grosvenor-street, W. c 3.
1872. \*ALLINGHAM, WILLIAM, F.R.C.S., 25, Grosvenor-street, W. c.
1860. ALTHAUS, JULIUS, M.D., 48, Harley-street, W. c 5, § 3.

1885. ANDERSON, JOHN, M.D., C.I.E., 9, Harley-street, W.  
 1889. ANDERSON, WILLIAM, F.R.C.S., 2, Harley-street, W.  
 1888. ANDREWES, FREDERICK WILLIAM, M.B., 15, Upper Brook-street, W.  
 1869. ARMITAGE, SAMUEL HARRIS TATHAM, M.D., 39, Grosvenor-street, W.  
 1873. ATKINSON, EDWARD, M.R.C.S., 16, Blenheim-terrace, Leeds.  
 1892. AYRES, CHARLES JAMES, M.D., 15, Grosvenor-road, Westminster, S.W.
1873. BAGSHAW, FREDERIC, M.D., 35, Warrior-square, St. Leonards-on-Sea. c.  
 1871. BAILEY, GEORGE HEWLETT, M.R.C.S., 9, Cavendish-place, W.  
 1892. BAILEY, HENRY FREDERICK, M.R.C.S., The Hollies, Lee-terrace, Lee,  
 S.E. *Councillor*.  
 1891. BAILY, PERCY J., M.B., County Asylum, Hanwell, W.  
 1876. \*BAKER, HENRY FRANCIS, F.R.C.S. Edin., 2, Mandeville-place,  
 Manchester-square, W. c.  
 1890. BAKER, WILLIAM HENRY, M.R.C.S., 40, Norfolk-terrace, Bayswater, W.  
 1891. BALL, JAMES BARRY, M.D., 54, Wimpole-street, W.  
 1881. BALLANCE, CHARLES ALFRED, M.S., 106, Harley-street, W. s 2, c 2,  
*Councillor*.  
 1884. BANKS, W. MITCHELL, F.R.C.S., 28, Rodney-street, Liverpool. o.  
 1859. BARNES, JOHN WICKHAM, F.R.C.S., 3, Bolt-court, E.C. s 2, VP, c 3.  
 1883. \*BARNES, ROBERT, M.D., *Honorary Fellow (q. v.)*.  
 1874. BARRETT, HOWARD, M.R.C.S., 49, Gordon-square, W.C.  
 1884. BARROW, ALBERT BOYCE, F.R.C.S., 37, Wimpole-street, W. c.  
 1886. BARWELL, RICHARD, F.R.C.S., 55, Wimpole-street, W.  
 1884. BATEMAN, FREDERICK AUGUSTUS NEWTON, M.R.C.S., 4, Charles-street,  
 St. James's-street, S.W.  
 1886. BATTERHAM, JOHN WILLIAMS, M.B., Bank House, Grand-parade, St.  
 Leonards-on-Sea.  
 1888. BATTLE, WILLIAM HENRY, F.R.C.S., 2, Mansfield-street, W.  
 1882. BEACH, FLETCHER, M.B., Two Elms, Sidcup, Kent. c.  
 1887. BEALE, EDWIN CLIFFORD, M.B., 23, Upper Berkeley-street, W.  
 1891. BEALE, PEYTON T. B., F.R.C.S., 61, Grosvenor-street, W.  
 1880. BEEVOR, CHARLES EDWARD, M.D., 33, Harley-street, W. s 2, c.  
*Trustee*.  
 1889. BEEVOR, SIR HUGH REEVE, Bart., M.D., King's College-chambers,  
 Strand, W.C.  
 1887. BENHAM, FREDERICK LUCAS, M.D., 93, Elizabeth-street, Eaton-square,  
 S.W.  
 1881. BENNET, ROBERT OTTIWELL-GIFFORD, M.D., Tankerville House, Park-  
 place, Buxton.  
 1883. BENNETT, WILLIAM HENRY, F.R.C.S., 1, Chesterfield-street, Mayfair, W.  
 1887. BERRY, JAMES, F.R.C.S., 60, Welbeck-street, W.  
 1893. BETTS, FREDERICK BERNARD, M.R.C.S., 63, The Chase, Clapham  
 Common, S.W.  
 1873. BEVERIDGE, JAMES SPOWART, M.R.C.P. Edin., Lochinver, Lairg, N.B.  
 1890. BIDWELL, LEONARD ARTHUR, F.R.C.S., 54, Harley-street, W.



1868. BIRD, GEORGE, M.D., 49, Welbeck-street.
1888. BIRD, MATTHEW MITCHELL, M.D., St. Mary's Hospital, W.
1850. \*BIRKETT, JOHN, F.R.C.S., 62, Green-street, Grosvenor-square, W.  
VP, C 6.
1883. BISS, CECIL YATES, M.D., 135, Harley-street, W.
1889. BISSHOPP, FRANCIS ROBERT BRYANT, M.B., Belvedere, Lonsdale-gardens, Tunbridge Wells.
1886. \*BLACK, WILLIAM GALL, F.R.C.S., 2, George-square, Edinburgh.
1885. BLAKE, JOHN FRENCH, Terrace House, Camberwell-green, S.E.
1881. BLAKER, WALTER CAMPBELL, Bognor, Sussex.
1888. BLANC, LEON, M.D., Aix les Bains, France.
1871. \*BLOXAM, JOHN ASTLEY, F.R.C.S., 75, Grosvenor-street, W. VP 2, S 2, C 3.
1867. BOND, THOMAS, F.R.C.S., 7, The Sanctuary, Westminster, S.W. C.
1879. BOTT, HENRY, M.R.C.S., Brentford, Middlesex.
1872. BOULTON, PERCY, M.D., 6, Seymour-street, Portman-square, W. C.
1886. BOURNS, NEWCOME WHITELAW, M.D., 449, Fulham-road, S.W.
1886. BOUSTEAD, ROBINSON, M.D. (Brigade Surgeon), 10, Palmeira-avenue, West Brighton.
1889. BOWLES, ROBERT LEAMON, M.D., 16, Upper Brook-street, W.
1883. BRADSHAW, JAMES DIXON, M.B.,
1868. BRAIDWOOD, PETER MURRAY, M.D., 2, Grosvenor-gardens, Willesden-green, N.W. F.M., 1877.
1869. BRAINE, FRANCIS WOODHOUSE, F.R.C.S., 56, Maddox-street, W.  
VP 2, S 2, C 3, SM.
1889. BRAINE, C. CARTER, F.R.C.S., 56, Maddox-street, W.
1876. BREWER, ALEXANDER HAMPTON, 136, Richmond-road, Dalston, E.
1873. BRIDGWATER, THOMAS, M.B., LL.D., Harrow, Middlesex. C.
1887. BRISTOWE, JOHN SYER, M.D., F.R.S., 13, Old Burlington-street, W.  
LL. *President*.
1893. BROADBENT, JOHN FRANCIS HARPIN, M.B., 84, Brook-street, W.
1862. BROADBENT, Sir WILLIAM HENRY, Bart., M.D., 84, Brook-street, W.  
P, VP, O, LL, C 4.
1890. BROOK, WILLIAM FREDERICK, F.R.C.S., Mount Pleasant, Swansea.
1879. BROOKFIELD, JOHN STORRS, M.D., 2, Devonshire-villas, Brondesbury, N.W.
1878. BROOKS, JOB EDWIN, 54, Mill-street, Ludlow, Salop.
1878. BROWN, ANDREW, M.D., Elton Villa, 1, Bartholomew-road, Kentish Town, N.W.
1871. BROWN, JOHN, St. Levan's, 28, Lavender-gardens, Lavender-hill, S.W.
1889. BROWNE, GEORGE BUCKSTON, M.R.C.S., 80, Wimpole-street, W.  
*Councillor*.
1871. BROWNE, Sir JAMES CRICHTON, M.D., F.R.S., 61, Carlisle-mansions, S.W. O, C.
1873. BROWNE, LENNOX, F.R.C.S. Edin., 15, Mansfield-street, Portland Place, W.
1887. BRUCE, JOHN MITCHELL, M.D., 70, Harley-street, W.

1873. BRUNJES, MARTIN, M.R.C.S., 33A, Gloucester-place, Bryanston-square, W.
1862. BRUNTON, JOHN, M.D., 21, Euston-road, N.W. VP, c 2.
1874. \*BRUNTON, THOMAS LAUDER, M.D., F.R.S., 10, Stratford-place, W. LL, VP, c 4, SM.
1850. \*BRYANT, THOMAS, F.R.C.S., 65, Grosvenor-street, W. P, VP, LL, s 2, c 4. *Trustee.*
1858. BUCHANAN, Sir GEORGE, M.D., F.R.S., 27, Woburn-square, W.C. P, LL, VP, O, c 3.
1883. BULL, WILLIAM HENRY, St. Oswald's House, Stony Stratford, Bucks.
1885. \*BUNNY, J. BRICE, M.R.C.S., Newbury, Berks.
1872. BURGER, ALEXANDER, M.D., 49, Finsbury-square, E.C.
1890. BUTLER, PATRICK, L.K.Q.C.P., 22, Duke-street, Portland-place, W.
1886. BUTLER-SMYTHE, ALBERT CHARLES, F.R.C.S., 76, Brook-street, W.
1872. BYAS, EDWARD HEGLEY, M.R.C.S., 10, Cambridge-gate, Regent's Park, N.W.
1886. CAHILL, JOHN, F.R.C.S., 12, Seville-street, Lowndes-square, Hyde Park, S.W. *Councillor.*
1892. CALDWELL, ROBERT, F.R.C.S., Surgeon Captain, care of Messrs. Holt and Co., 17, Whitehall-place, S.W.
1893. CALEY, HENRY ALBERT, M.D., 19, Lower Seymour-street, W.
1891. CALVERT, JAMES, M.D., 36, Queen Anne-street, W.
1888. CAMPBELL, CHARLES M., M.D., *Travelling.*
1890. CARDEW, HARRY WARNELL DENTON, M.R.C.S., 53, Harley-street, W.
1892. CARGILL, LIONEL VERNON, F.R.C.S., Royal Eye Hospital, St. George's-circus, S.E.
1892. CARLESS, ALBERT, F.R.C.S., 10, Welbeck-street, W.
1889. CARNALL, EDWARD, M.R.C.S., 9, Gerrard-street, W.
1882. CARPENTER, ARTHUR BRISTOWE, M.B., 34, Dingwall-road, Croydon.
1889. CARR, JOHN WALTER, M.D., 40, Bloomsbury-square, W.C.
1871. CARTER, ROBERT BRUDENELL, F.R.C.S., 27, Queen Anne-street, W., P, VP, O, LL, c 4.
1889. CARTWRIGHT, ALEXANDER, M.R.C.S., 32, Old Burlington-street, W.
1876. CARTWRIGHT, S. HAMILTON, 45, Albert-gate, Hyde-park, W.
1878. CASSIDY, JOSEPH LAMONT, M.D., 44, Harley-street, W.
1876. \*CATHCART, SAMUEL, M.R.C.P. Edin., Prudhoe House, High-road, Tottenham, E.
1889. CAUTLEY, EDMUND, M.B., 15, Upper Brook-street, W.
1882. CAVAFY, JOHN, M.D., 2, Upper Berkeley-street, W. c 3.
1891. CHAPLIN, T. H. ARNOLD, M.B., 24, Finsbury-circus, E.C.
1867. CHAPMAN, JOHN, M.D., Avenue de l'Opera 31, Paris.
1885. CHASSEAUD, WILLIAM, M.D., Smyrna, Asia Minor.
1889. CHEYNE, WATSON, F.R.C.S., 75, Harley-street, W. c.
1877. \*CHISHOLM, EDWIN, M.D., Sydney, New South Wales.
1871. CHURTON, THOMAS, M.D., 35, Park-square, Leeds. c.

1854. CLARK, Sir ANDREW, Bart., M.D., F.R.S., 16, Cavendish-square, W.  
President of the Royal College of Physicians. P, VP, LL, C 5, § 6.
1875. CLARK, ANDREW, F.R.C.S., 71, Harley-street, W.
1873. CLARKE, THOMAS KILNER, F.R.C.S., 66, John William-street, Huddersfield.
1883. CLARKE, WILLIAM BRUCE, F.R.C.S., 46, Harley-street, W. c.
1879. \*CLUTTON, HENRY HUGH, F.R.C.S., 2, Portland-place, W. c 2.
1849. \*COCKLE, JOHN, M.D., 5, Suffolk-place, Pall Mall, S.W. P, VP, O, L 3,  
C 3, SM.
1893. COLE, ROBERT HENRY, M.B., Moorcroft, Hillingdon, Middlesex.
1887. COLLIER, WILLIAM, M.D., 62, High-street, Oxford. *Councillor*.
1893. COLLUM, ARCHIE TILLYER, F.R.C.S., Charing Cross Hospital, W.C.
1892. COLMAN, WALTER S., M.D., 41, Torrington-square, W.C.
1871. COOK, JOHN, M.D., 1, Nottingham-terrace, Regent's Park, N.W.
1862. COOPER, ALFRED, F.R.C.S., 9, Henrietta-street, Cavendish-square, W.  
C 3, VP.
1888. COOPER, ARTHUR, 20, Old Burlington-street, W.
1872. CORFIELD, WILLIAM HENRY, M.D., 19, Savile-row. c.
1892. COTTERELL, EDWARD, F.R.C.S., 5, West Halkin-street, Belgrave-square, S.W.
1891. COUMBE, JOHN BATTEN, M.D., Wargrave, Henley-on-Thames.
1879. COUPLAND, SIDNEY, M.D., 16, Queen Anne-street, W. c 2.
1889. COURTNEY, GUY BUDD, M.B., 47, Seymour-street, W.
1874. CRAIGIE, JOHN HAMILTON, 13, Savile-row, W. c.
1873. CRAVEN, ROBERT MARTIN, F.R.C.S., J.P., 14, Albion-street, Hull.
1889. CRAWFORD, JAMES, M.D., Grosvenor-mansions, 80, Victoria-street, S.W.
1881. CRIPPS, WILLIAM HARRISON, F.R.C.S., 2, Stratford-place, W. c 2.
1880. CRITCHETT, GEORGE ANDERSON, F.R.C.S. Edin., 21, Harley-street, W.
1880. CROCKER, HENRY RADCLIFFE, M.D., 121, Harley-street, c 3.
1881. CROSS, FRANCIS RICHARDSON, F.R.C.S., Worcester House, Clifton, Bristol.
1890. CULLINGWORTH, CHARLES JAMES, M.D., 46, Brook-street, W. c 2.  
*Councillor*.
1874. CUMBERBATCH, ALPHONSO ELKIN, F.R.C.S., 17, Queen Anne-street, W. c 2.
1892. DA COSTA, FRANCIS XAVIER, F.R.C.S., Charing Cross Hospital, W.C.
1871. DALBY, Sir WILLIAM BARTLETT, F.R.C.S., 18, Savile-row, W. c.
1864. DALE, GEORGE CORNELIUS, M.D., 13, Nightingale Park-crescent, Wandsworth Common, S.W.
1881. DALLAWAY, DENNIS JOSEPH WILLIAM, L.R.C.P. Edin., 5, Duchess-street, Portland-place, W.
1873. DALY, OWEN, M.D., J.P., 23, Albion-street, Hull.
1885. DAVIES-COLLEY, JOHN NEVILLE COLLEY, F.R.C.S., 36, Harley-street, W. c 3.
1890. DAVIS, HENRY, M.R.C.S., 60, Queen Anne-street, W.



1889. \*DAVISON, JAMES, M.D., Walderslow, Pournemouth.
1880. DAVSON, SMITH HOUSTON, M.D., Campden Villa, 203, Maida-vale, W. c 3.
1868. \*DAVY, RICHARD, F.R.C.S., F.R.S.E., 33, Welbeck-street, W. VP, s 2,  
SM, § 2.
1876. DAWES, RICHARD ST. MARK, M.R.C.S., Gawler, South Australia.
1880. DAWSON, YELVERTON, M.D., Heathland, Southborne-on-Sea, Christ-  
church, Hants.
1867. DAY, WILLIAM HENRY, M.D., 10, Manchester-square, W. c 3.
1883. DENT, CLINTON THOMAS, F.R.C.S., 61, Brook-street, Grosvenor-square,  
W. c 3.
1891. DIVER, EBENEZER, M.D., Yately House, Kenley, Surrey.
1885. DODD, HENRY WORK, F.R.C.S., 136, Harley-street, W.
1882. DOLAN, THOMAS MICHAEL, M.D., Horton House, Halifax. FM 1882.
1881. DORAN, ALBAN HENRY GRIFFITHS, F.R.C.S., 9, Granville-place, W. c 3.
1890. DOUGLAS, WILLIAM, M.D., Dalkeith House, 7, Clarendon-place,  
Leamington Spa.
1871. DOWSE, THOMAS STRETCH, M.D., 14, Welbeck-street, W. § 3, c 3.
1877. DREW, JOHN HENRY, M.R.C.S., 38, Eastbourne-terrace, Hyde Park, W.  
c 6.
1881. DREWITT, FREDERIC GEORGE DAWTREY, M.D., 2, Manchester-square, W.
1874. DRYSDALE, CHARLES ROBERT, M.D., 23, Sackville-street, W.
1886. DUCKWORTH, Sir DYCE, M.D., 11, Grafton-street, Piccadilly, W.
1848. \*DUNCAN, JAMES, M.B., 8, Henrietta-street, Covent Garden, W.C.
1884. DUNCAN, WILLIAM, M.D., 6, Harley-street, W.
1873. \*DURHAM, ARTHUR EDWARD, F.R.C.S., 82, Brook-street, W. P, O, C.  
*Treasurer.*
1884. DURHAM, FREDERICK, F.R.C.S., 82, Brook-street, W.
1891. EASTES, THOMAS, M.D., 3, Shakespeare-terrace, Folkestone.
1893. ECCLES, ARTHUR SYMONS, M.B., 23, Hertford-street, Mayfair, W.
1892. EDDOWES, ALFRED, M.D., 25, Old Burlington-street, W.
1860. EDMUNDS, JAMES, M.D., 29, Dover-street, W.
1880. EDWARDS, FREDERICK SWINFORD, F.R.C.S., 55, Harley-street, W.  
*Councillor.*
1868. ELLIOTT, GEORGE FREDERICK, M.D., 1, Albion-street, Hull.
1882. ELLIOTT, THOMAS, M.D., Monson-place, Tunbridge Wells.
1889. EMBLETON, DENNIS CAWOOD, M.R.C.S., St. Wilfrid's, Bournemouth.
1883. EMOND, E., M.D., 113, Boulevard Beaumarchais, Paris.
1883. ENGLISH, EDGAR, M.R.C.S., High-street, Mexborough, near Rotherham.
1880. ENGLISH, THOMAS JOHNSTON, M.D., 128, Fulham-road, S.W.
1889. ESLER, ROBERT, M.D., 4, Queen's-road, Peckham, S.E.
1891. EUAN-SMITH, EUAN McLAURIN, M.R.C.S., 253, Cromwell-road, S.W.
1883. EWART, JOSEPH, M.D., J.P., Retired Dep. Surgeon-General, Bengal  
Army, Montpellier Hall, Brighton. c.
1877. EWART, WILLIAM, M.D., 33, Curzon-street, Mayfair, W. c.

1889. FAIRBANK, FREDERICK ROYSTON, M.D., 59, Warrior-square, St. Leonards-on-Sea.
1884. FARDON, EDWARD ASHBY, M.R.C.S., Middlesex Hospital, W.
1873. FAYRER, Sir JOSEPH, K.C.S.I., LL.D., M.D., F.R.S., 53, Wimpole-street, W. p, vp, ll, sm, o, c.
1884. FENTON, FREDERICK ENOS, F.R.C.S., Langstone, Ealing, W.
1888. FENWICK, BEDFORD, M.D., 20, Upper Wimpole-street, W.
1885. FENWICK, EDWIN HURRY, F.R.C.S., 5, Old Burlington-street, W.
1887. FERRIER, DAVID, M.D. Edin., F.R.S., 34, Cavendish-square, W.
1878. FIELD, GEORGE, F.R.C.S., 34, Wimpole-street, W. c.
1883. FINLAY, DAVID WHITE, M.D., 2, Queen's-terrace, Aberdeen. c 2.
1876. FISHER, FREDERIC RICHARD, F.R.C.S., 17, Wimpole-street, W.
1884. FLINT, ARTHUR, M.D., Westgate Lodge, Westgate-on-Sea.
1878. \*FONMARTIN, HENRY DE, M.D., 1, Anchor-gate-terrace, Portsea, Hants.
1884. FOTHERBY, HENRY ARTHUR, 17, Scarsdale-terrace, Cheniston-gardens, Kensington, W.
1879. FOWLER, JAMES KINGSTON, M.D., 35, Clarges-street, Mayfair, W. s 2, c.
1873. FOX, ARTHUR EDWARD WELLINGTON, M.B., C.M., 16, Gay-street, Bath. c.
1887. FOX, FORTESCUE, M.D., Strathpeffer Spa, Ross-shire.
1871. FOX, FRANCIS, M.R.C.S., 68, Wimpole-street, W. c 3.
1885. FOX, R. HINGSTON, M.D., 23, Finsbury-square, E.C.
1879. FOX, THOMAS COLCOTT, M.B., 14, Harley-street, W. s 2. c. *Councillor*.
1887. FRAZER, ROBERT FAIR, 185, Lavender-hill, New Wandsworth, S.W.
1868. FREER, ALFRED, J.P., Stourbridge, Worcestershire.
1886. FRITH, BAPTIST GAMBLE, M.B., 29, Cornwallis-gardens, Hastings.
1884. FROST, WILLIAM ADAMS, F.R.C.S., 17, Queen Anne-street, W. c 2.
1883. GABBETT, HENRY SINGER, M.D., 8, Chiswick-place, Eastbourne.
1862. GANT, FREDERICK JAMES, F.R.C.S., 16, Connaught-square, W. p, vp 2, ll, o, c 3.
1847. \*GARROD, Sir ALFRED BARING, M.D., F.R.S., 10, Harley-street, W. p, vp 2, ll, o, c 9.
1887. GARROD, ARCHIBALD EDWARD, M.D., 9, Chandos-street, Cavendish-square, W. *Councillor*.
1891. GASTER, AUGHEL, M.D., 34, Warwick-road, Maida-vale, W.
1887. GAY, JOHN, 119, Upper Richmond-road, Putney, S.W.
1879. GIBBES, HENEAGE, M.D., The University, Michigan, U.S.A.
1856. GIBBON, SEPTIMUS, M.B., 39, Oxford-terrace, W.
1882. GIBBONS, ROBERT ALEXANDER, M.D., 29, Cadogan-place, S.W.
1881. GIFFARD, DOUGLAS W., M.R.C.S., 5, Pavilion-parade, Brighton.
1867. GILL, WILLIAM, M.R.C.S., 11, Russell-square, W.C. c.
1869. GODSON, CLEMENT, M.D., 9, Grosvenor-street, W. vp, c 3, s 2, sm.

1873. GOODSALL, DAVID HENRY, F.R.C.S., 17, Devonshire-place, Portland-place, W. c, s 2, SM, CFC 7. *Vice-President ; Chairman, House and Finance Committee.*
1892. GORDON, ROBERT JOHN, M.B.
1880. GOUDE, HERBERT, M.D., Smallpox Hospital, Highgate-hill, N.
1878. \*GOULD, ALFRED PEARCE, M.S., 10, Queen Anne-street, W. s 2, c 3.
1876. GOWERS, WILLIAM RICHARD, M.D., F.R.S., 50, Queen Anne-street, W. VP, C, SM, LL. FM. 1893.
1874. GOWLLAND, PETER YEAMES, F.R.C.S., 82, Gloucester-terrace, Hyde-park, W.
1887. GRANT, JAMES EDWARD RONEY, 2, Charing Cross-chambers, Duke-street, Adelphi, W.C.
1881. GREEN, THOMAS HENRY, M.D., 74, Wimpole-street, W. c 2. VP.
1868. GREGSON, GEORGE, M.R.C.S., 63, Harley-street.
1886. GREVES, EDWIN HYL, M.D., Rodney House, Bournemouth.
1873. GRIEVE, ROBERT, M.D., British Guiana.
1884. GRIFFITH, DAVID CHARLES BALLINGER, M.R.C.P. Edin., 3, Lansdowne-place, Brighton.
1875. GRIFFITH, G. DE GORREQUER, M.R.C.S., 34, St. George's-square, S.W.
1885. GRIFFITHS, CHARLES THOMAS, L.R.C.P., 206, Lozells-road, Birmingham.
1884. GRIFFITHS, HERBERT TYRRELL, M.B., 5, Kensington-square, W.
1880. GRISTOCK, WILLIAM, M.D. Lond., 6, Finchley-road, N.W.
1893. GUBB, ALFRED SAMUEL, M.D., 29, Gower-street, W.C.
1891. GUTHRIE, LEONARD G., M.B., 24, Upper George-street, Bryanston-square, W.
1886. HABERSHON, S. HERBERT, M.D., 70, Brook-street, Grosvenor-square, W.
1891. HADLEY, WILFRED J., M.B., 16, Wimpole-street, W.
1887. HAIG, ALEXANDER, M.B., 7, Brook-street, W.
1884. HAIRSINE, HUDSON, Roose House, Upper Tooting, S.W.
1881. HALL, CHARLES ROSS, M.R.C.S., Hatfield, Herts.
1874. \*HALL, FRANCIS DE HAVILLAND, M.D., 47, Wimpole-street, W. c 4, s 2, SM. *Vice-President.*
1885. HALPIN, RICHARD FREDERICH BESTALL, Arklow, co. Wicklow, Ireland.
1881. HAMES, GEORGE HENRY, F.R.C.S., 29, Hertford-street, Mayfair, W.
1892. HAMILTON, JOHN BUTLER, M.D., Surgeon-Colonel, 28, Cockspur-street, Charing Cross, S.W.
1879. HAMILTON, SETON GUTHRIE, Surgeon-Captain, 1st Life Guards.
1891. HANDFIELD-JONES, MONTAGU, M.D., 35, Cavendish-square, W.
1887. HANDFORD, HENRY, M.D. Edin., 14, Regent-street, Nottingham.
1850. \*HARE, CHARLES JOHN, M.D., *Honorary Fellow (q. v.).*
1888. HARE, HOBART AMORY, 117, South Twenty-second-street, Philadelphia. FM. 1888.
1891. HAROLD, JOHN PATRICK, M.R.C.S., 91, Harley-street, W.



1882. HARPER, GERALD SAMUEL, M.B., 40, Curzon-street, Mayfair, W.  
*Councillor.*
1871. HARRIS, CHARLES JAMES, 4, Kilburn Priory, N.W.
1871. HARRISON, REGINALD, F.R.C.S., 6, Lower Berkeley-street, Portman-square. VP 2, LL, c 1.
1883. \*HARTRIDGE, GUSTAVUS, F.R.C.S., 65, Green-street, Grosvenor-square, W.
1864. HARVEY, JOHN ALEXANDER, 35, Princes-square, Bayswater, W.
1882. HARVEY, JOHN STEPHENSON SELWYN, M.D., 1, Astwood-road, Cromwell-road, S.W.
1882. HASLAM, WILLIAM FREDERICK, F.R.C.S., 33, Paradise-street, Birmingham. c 3.
1852. \*HAWARD, EDWIN, M.D., 34A, Gloucester-place, W.
1883. HAWKEN, CHARLES ST. AUBYN, 20, North-terrace, Wandsworth, S.W.
1889. HAWKINS, FRANCIS HENRY, M.B., 59, Wimpole-street, W.
1890. HEBB, FREDERICK THEODORE, M.R.C.S., 7, Milner-terrace, Cadogan-square, S.W.
1884. HENSMAN, ARTHUR, F.R.C.S., 31, Harley-street, W.
1891. HENSMAN, FRANK, M.R.C.S., Surgeon-Major, 1st Life Guards.
1883. HERMAN, GEORGE ERNEST, M.B., 20, Harley-street, W.
1879. HERON, GEORGE ALLAN, M.D., 57, Harley-street, Cavendish-square, W.  
c 2. *Councillor.*
1886. HERRINGHAM, WILMOT PARKER, M.B., 13, Upper Wimpole-street, W.
1883. HERSHELL, GEORGE A., M.D., 25, Queen Anne-street, W.
1883. HEWITT, FREDERICK WILLIAM, M.D., 10, George-street, Hanover-square, W.
1876. HEYCOCK, FRANCIS RAWORTH, C.M., 26, Upper Wimpole-street, W.
1872. HICKS, JOHN BRAXTON, M.D., F.R.S., 34, George-street, Hanover-square. c.
1892. HILL, WILLIAM, M.D., 24, Wimpole-street, W.
1873. HOBSON, WILLIAM HENRY, M.R.C.S., Great Berkhamstead, Herts.
1879. HOGG, ARTHUR JOHN, M.R.C.S., Leslie Lodge, Haven-green, Ealing, W.
1892. HOGG, FREDERICK STAPLETON D., M.R.C.S., 9, Nottingham-terrace, N.W.
1884. HOLLAND, CHARLES EDWARD, M.B., 44, Warwick-road, Maida-vale, W.
1888. HOLM, JOHN, F.R.C.S. Edin., 13, Stratford-place, W.
1868. HOLMAN, CONSTANTINE, M.D., 26, Gloucester-place, Portman-square, W. c 4.
1881. HOOD, DONALD WILLIAM CHARLES, M.D., 43, Green-street, W. c 2.
1879. HOOKHAM, PAUL (address uncommunicated).
1893. HORROCKS, HERBERT, M.D., N.E. Hospital for Children, Hackney-road, N.E.
1883. \*HOVELL, T. MARK, F.R.C.S. Edin., 105, Harley-street, W.
1886. HUDDART, CUTHBERT HENRY COOKE, M.B., Shoyswell Manor, Etchingham, Sussex.
1885. HUDSON, CHARLES ELLIOTT LEOPOLD BARTON, F.R.C.S., 6, Chandos-street, W.

1890. HUGHES, EDGAR, F.R.C.S., 91, Onslow-gardens, South Kensington, S.W.  
 1864. HUME, FREDERICK HENRY, M.D., 53, Devonshire-street, Islington, N.  
 1884. HUNTER, Sir GUYER, M.D., 21, Norfolk-crescent, Hyde Park, W.  
 1889. HUNTER, WILLIAM, M.D., 54, Harley-street, W.  
 1881. HUTCHINSON, JONATHAN, F.R.C.S., F.R.S., 15, Cavendish-square. P, LL,  
     c 4, o. *Councillor*.  
 1892. HUTCHINSON, JONATHAN, Jun., F.R.C.S., 1, Park-crescent, Portland-  
     place, W.  
 1875. HUTCHINSON, SAMUEL JOHN, M.R.C.S., 64, Brook-street, W.  
 1889. I'ANSON, WILLIAM ANDREW, Denton Hall, Newcastle-on-Tyne.  
 1891. ISAAC, GEORGE WASHINGTON, M.B., 75, Gower-street, W.C.  
 1884. \*JACKSON, FREDERICK WILLIAM, M.D., Yorkgate House, Broadstairs.  
 1885. JACKSON, JAMES, M.R.C.S., 15, Huntingdon-street, Barnsbury, N.  
 1868. JACKSON, JOHN HUGHLINGS, M.D., F.R.S., 3, Manchester-square, W.  
     P, VP, O, C 5.  
 1874. JAGIELSKI, VICTOR APOLLINARIS, M.D., 54, York-terrace, Regent's  
     Park, N.W.  
 1882. JAMES, JOSEPH BRINDLEY, M.R.C.S., 47, Jamaica-road, Bermondsey,  
     S.E.  
 1887. JAMISON, ARTHUR ANDREW, M.D., 18, Lowndes-street, Belgrave-  
     square, S.W.  
 1884. JENNINGS, CHARLES EGERTON, F.R.C.S., 48, Seymour-street, W.  
 1886. JERVIS, ARTHUR, M.R.C.S., Seamen's Hospital, Greenwich, S.E.  
 1883. JESSETT, FREDERICK BOWREMAN, F.R.C.S., 1, Buckingham Palace-  
     mansions, S.W.  
 1883. JESSOP, WALTER HAMILTON, F.R.C.S., 73, Harley-street, W.  
 1893. JOHNSTON, GEORGE FRANCIS, M.D., 6, Manchester-square, W.  
 1886. JOHNSTON, JAMES, M.D., 11, Chester-place, Hyde Park-square, W.  
 1888. JONES, ARTHUR HENRY, M.D., 45, Sheep-street, Northampton.  
 1890. JONES, H. MACNAUGHTON, M.D., 141, Harley-street, Cavendish-square,  
     W.  
 1888. JONES, JOHN TALFOURD, M.B., Rosebank, South-terrace, Eastbourne,  
     Sussex.  
 1892. \*JONES, ROBERT, F.R.C.S., 11, Nelson-street, Liverpool.  
 1881. JONES, THOMAS WILLIAM CARMALT, F.R.C.S. Edin., 6, Westbourne-  
     street, Hyde Park, W.  
 1893. JOULE, JOHN SAMUEL, M.D., 32, Maida-hill West, W.  
 1877. JULER, HENRY EDWARD, F.R.C.S., 23, Cavendish-square, W.  
 1889. KAUFFMANN, OTTO JACKSON, M.D., Queen's Hospital, Birmingham.  
 1874. KAVANAGH, PATRICK, M.D., 81, Marine-parade, Brighton.  
 1891. KEEGAN, DENIS FRANCIS, M.D., Surgeon-Major, The Residency, Indore,  
     Central India.  
 1884. KEETLEY, CHARLES BELL, F.R.C.S., 56, Grosvenor-street, W. c 3.

1892. KEIFFENHEIM-TRUBRIDGE, LUIGI W. A., M.D., 4, Cliveden-place, Eaton-square, S.W.
1847. \*KELLOCK, WILLIAM BERRY, M.D., 94, Stamford-hill, N.
1890. KELLY, AUGUSTIN BERNARD, M.R.C.S., 82, Park-street, Grosvenor-square, W.
1891. KELSON, WILLIAM HENRY, M.D., 46, Watling-street, E.C.
1883. KEMP, JOHN ROBERT, M.R.C.S., 101, Jermyn-street, S.W.
1890. KER, HUGH RICHARD, F.R.C.S. Edin., Devonshire Cottage, Balham Hill, S.W.
1884. KERR, NORMAN, M.D., 42, Grove-road, N.W.
1881. KESER, JEAN SAMUEL, M.D., 11, Harley-street, W. c 2, § 3.
1876. KEY, AUGUSTUS COOPER, M.D., 30, Wilton-place, S.W. c. *Councillor*.
1886. KIDD, PERCY, M.D., 60, Brook-street, W. c 2.
1889. KIRKHAM, FREDERICK WILLIAM, Downham Market, Norfolk.
1883. KNAPTON, GEORGE, Cliveden House, 57, Queen Anne-street, W.
1875. KNOX, JOHN, M.D., Bethnal Green Infirmary, E.
1889. LAKE, RICHARD, F.R.C.S., 46, Seymour-street, W.
1868. LAKE, WILLIAM CHARLES, M.D., Teignmouth, Devon.
1872. LANGDON-DOWN, JOHN L. H., M.D., J.P., 81, Harley-street, W. v.p, LL.
1881. LANGTON, JOHN, F.R.C.S., 62, Harley-street, W. c 2.
1882. LARKIN, F. COLET, M.B., Kingsbridge House, Avenue-road, East Cliff, Ramsgate.
1890. LAW, EDWARD, M.D. Edin., 35, Harley-street, W.
1890. LAWRIE, EDWARD, M.B. Edin., Surgeon-Major, Bengal Army, The Residency, Hyderabad.
1858. LAWSON, GEORGE, F.R.C.S., 12, Harley-street, W. vp 2, c 3.
1891. LAZARUS-BARLOW, WALTER SYDNEY, M.B., The Acacias, Chesterton, Cambridge.
1893. LEE, E. SAMUEL, M.D., 31, Pevensey-road, St. Leonards-on-Sea.
1887. LEGGATT, CHARLES ASHLEY SCOTT, M.D., 2, Walton-place, S.W.
1858. LEMON, OLIVER, M.R.C.S., 12, The Grange, Highbury, N.
1886. LEWERS, ARTHUR HAMILTON NICHOLSON, M.D. Lond., 60, Wimpole-street, W.
1867. LICHTENBERG, GEORGE, M.D., 47, Finsbury-square. c 2.
1893. LIGHT, EDWIN MELLOP, M.B., 2, Wilton-place, S.W.
1878. LISTER, Sir JOSEPH, Bart., D.C.L., LL.D., F.R.C.S., F.R.S., 12, Park-crescent, Portland-place. o.
1890. LITHGOW, ROBERT ALEXANDER DOUGLAS, M.D., LL.D., 27A, Lowndes-street, S.W.
1889. LITTLE, ERNEST MUIRHEAD, F.R.C.S., 40, Seymour-street, Portman-square, W.
1889. LITTLE, FLETCHER, M.D., 32, Harley-street, W.
1887. LLOYD, ROBERT HODGENS, M.D., Brook House, Kennington-road, S.E.
1886. LLOYD, SAMUEL, M.D., 4, High-street, Bloomsbury, W.C.



1878. LOCKWOOD, CHARLES BARRETT, F.R.C.S., 19, Upper Berkeley-street, W. c. *Honorary Secretary*.
1873. LOE, JAMES SCARBOROUGH, 26, Woodhouse-lane, Leeds.
1881. LORIMER, G., M.D., 9, Terrace-road, Buxton, Derbyshire.
1868. LOWE, JOHN, M.D., J.P., 4, Gloucester-place, Portman-square, W. c 3.
1868. \*LUND, EDWARD, F.R.C.S., 22, St. John's-street, Manchester. o, c 3.
1889. LUNN, HENRY SIMPSON, M.D., 5, Endsleigh-gardens, N.W.
1889. LUSH, PERCY, M.B., 4, Maresfield-gardens, Hampstead, N.W.
1884. MACBRYAN, HENRY CRAWFORD, Kingsdown House, Box, Wilts.
1871. MACCORMAC, Sir WILLIAM, F.R.C.S., 13, Harley-street, W. p, vp, § 2, c 4, o.
1882. MACKELLAR, ALEXANDER OBERLIN, F.R.C.S., 79, Wimpole-street, W.
1880. MACKENZIE, STEPHEN, M.D., 18, Cavendish-square, W. vp 2, c 3, LL. *Councillor*.
1881. MACLAGAN, THOMAS JOHN, M.D., 9, Cadogan-place, S.W. c 3.
1861. MACLAREN, ALEXANDER CONNELL, 60, Harley-street, W.
1891. MACLEAN, ALLAN, Harpenden Hall, Herts.
1887. MACREADY, JONATHAN FOSTER CHRISTIAN HORACE, F.R.C.S., 51, Queen Anne-street, W.
1883. MADDICK, EDMUND DISTIN, F.R.C.S. Edin., 2, Chandos-street, Cavendish-square, W.
1885. MAGUIRE, ROBERT, M.D., 4, Seymour-street, W. c 2.
1878. MAIR, ROBERT SLATER, M.D., 28, Ledbury-road, Bayswater, W.
1890. MALCOLM, JOHN DAVID, F.R.C.S. Edin., 13, Portman-street, Portman-square, W.
1893. MALCOLM, WILLIAM A., M.B., Oak House, 421, Holloway-road, N.
1887. MANTLE, ALFRED, M.D., Savile-place, Halifax.
1888. MAPOTHER, EDWARD DILLON, M.D., 32, Cavendish-square, W. *Councillor*.
1891. MARSH, HOWARD, F.R.C.S., 30, Bruton-street, W.
1892. MARSHALL, ARTHUR LUMSDEN, M.B., 56, Rectory-road, N.
1873. MARSHALL, EDWARD, M.R.C.S., Mitcham, Surrey.
1869. MARSHALL, WILLIAM, M.D., Torrieburn, Barnes, S.W.
1864. MARSHALL, WILLIAM GURSLAVE, F.R.C.S., 72, Bromfelde-road, Clapham, S.W.
1889. MARTIN, JOHN MICHAEL HARDING, M.D., Arnheim, Blackburn, Lancashire.
1890. MARTIN, SIDNEY, M.D., 10, Mansfield-street, W.
1884. MATHESON, FARQUHAR, M.B., 11, Soho-square, W.
1891. MAUDE, ARTHUR, M.R.C.S., Winterton House, Westerham, Kent.
1892. MAUNSELL, H. WIDENHAM, M.D., 37, Stanhope-gardens, Queen's-gate, S.W.
1892. MAY, CHICHESTER GOULD, M.D., 26, Walton-street, Pont-street, S.W.
1891. MAY, WILLIAM PAGE, M.D., 38, Weymouth-street, W.

1885. McCONNEL, HENRY WILSON, M.B., Litchdon, Barnstaple, Devon.
1885. McGEAGH, THOMAS EDWIN FOSTER, M.D., 23, New Cavendish-street, W.
1873. McHARDY, MALCOLM MACDONALD, F.R.C.S. Edin., 5, Savile-row, W.
1884. MEREDITH, WILLIAM APPLETON, C.M., 21, Manchester-square, W.
1864. MIDDLEMIST, ROBERT PERCY, M.R.C.S., 6, Devonport-street, Hyde Park, W. c 4.
1882. MILLS, JOSEPH, M.R.C.S., 28, Queen Anne-street, Cavendish-square, W.
1883. MONEY, ANGEL, M.D. (*travelling*). c.
1883. MOORE, THOMAS, F.R.C.S., 6, Lee-terrace, Blackheath, S.E.
1883. MORGAN, JOHN HAMMOND, F.R.C.S., 68, Grosvenor-street, W. s 2, c.
1893. MORISON, ALEXANDER, M.D., 14, Upper Berkeley-street, W.
1871. MORLEY, ALEXANDER, 42, Albemarle-street, W.
1881. MORRIS, HENRY, F.R.C.S., 8, Cavendish-square, W. c.
1878. MORRIS, MALCOLM ALEXANDER, F.R.C.S. Edin., 8, Harley-street, W. c 2.
1882. MORTON, ANDREW STANFORD, F.R.C.S., 26, Weymouth-street, Portland-place, W.
1884. \*MOULLIN, CHARLES WILLIAM MANSELL, F.R.C.S., 69, Wimpole-street, W.
1878. MUMFORD, WILLIAM LUGAR, M.D., 12, Suffolk-street, Pall-mall, S.W.
1893. MURPHY, GEORGE WYNDHAM, M.B., J.P., 98, Gloucester-crescent, Hyde Park, W.
1886. MURPHY, JAMES, M.D., Holly House, Sunderland.
1884. MURRAY, FRED., M.B., Durbanville, Cape Colony, South Africa.
1890. MURRAY, GEORGE, M.R.C.S., 34, Wimpole-street, Cavendish-square, W.
1886. MURRAY, HUBERT MONTAGUE, M.D., 27, Savile-row, W. *Councillor*.
1879. MURRELL, WILLIAM, M.D., 17, Welbeck-street, W.
1885. MYERS, ARTHUR THOMAS, M.D., 2, Manchester-square, W.
1893. NAPIER, ALEXANDER DISNEY LEITH, M.D., 67, Grosvenor-street, W.
1877. NESBITT, DAWSON, M.D., 1, Norfolk-square, Hyde Park, W.
1876. NEWHAM, JAMES, 80, Gloucester-place, W.
1889. \*NIAS, J. BALDWIN, M.B., 40, Brook-street, Grosvenor-square, W.
1880. NIX, EDWARD JAMES, M.D., 11, Weymouth-street, W.
1887. OAKLEY, ADAM ROBERT HAMILTON, L.R.C.P., Treath, Hornchurch, Essex.
1885. OGILVIE, LESLIE, M.B., 46, Welbeck-street, W.
1884. OGLE, CHARLES JOHN, 1, Cavendish-place, W.
1884. OLIVER, GEORGE, M.D., West End Park, Harrogate.
1892. OPENSHAW, THOMAS HORROCKS, F.R.C.S., 16, Wimpole-street, W.
1875. ORD, WILLIAM MILLER, M.D., 37, Upper Brook-street, W. p, c 4. *Orator*.
1892. ORD, WILLIAM WALLIS, M.D., 2, Queen-street, Mayfair, W.

## XXXVIII

1887. ORMEROD, JOSEPH ARDERNE, M.D., 25, Upper Wimpole-street, W. c.
1889. ORTON, GEORGE HUNT, M.B., 1A, Campden Hill-road, Kensington, W.
1884. ORWIN, ARTHUR WIGELSWORTH, M.D., 15, Weymouth-street, Portland-place, W.
1880. OSWALD, JAMES WADDELL JEFFREYS, M.D., 245, Kennington-road, S.E.
1883. OWEN, CHARLES J. RAYLEY, 14, Devonshire-terrace, Hyde Park, W.
1878. \*OWEN, EDMUND, F.R.C.S., 64, Great Cumberland-place, W. vp 2, c 3, s 2, SM, LL. *Trustee*.
1881. OWEN, ISAMBARD, M.D., 40, Curzon-street, Mayfair, W. s 2, c 4.
1886. PAGET, STEPHEN, F.R.C.S., 57, Wimpole-street, W. c 2.
1880. PALMER, FREDERICK STEPHEN, M.D., Compton Lodge, East Sheen, S.W.
1882. PALMER, WILLIAM PITT, M.B., 17, Belgrave-terrace, Torquay.
1877. \*PARAMORE, RICHARD, M.D., 2, Gordon-square, W.C.
1867. PARKINSON, GEORGE, 50, Brook-street, Grosvenor-square, W.
1881. PARROTT, EDWARD JOHN, M.R.C.S., The Thorn, Hayes, Middlesex.
1871. PARSONS, FRANCIS HENRY, M.D., "The Hurst," West Worthing.
1885. PASTEUR, WILLIAM, M.D., 4, Chandos-street, Cavendish-square, W. *Honorary Secretary*.
1872. PATTEN, CHARLES ARTHUR, M.R.C.S., Marpool House, Ealing, W.
1891. PATTERSON, CHARLES SUMNER, M.B., 353, City-road, E.C.
1890. PATTISON, EDWARD SETON, M.R.C.S., Granville House, Fulham-park, S.W.
1861. PAUL, JOHN HAYBALL, M.D., Camberwell House, Camberwell, S.E. c 6.
1854. PAVY, FREDERICK WILLIAM, M.D., F.R.S., 35, Grosvenor-street, W. vp, LL, C.
1881. \*PEACEY, WILLIAM, M.D., 11, Breakspears-road, Brockley, S.E.
1883. PECK, EDWARD GEORGE, M.A., Queensbury, Bradford, Yorks.
1871. PEDLER, GEORGE HENRY, M.R.C.S., 6, Trevor-terrace, Knightsbridge, S.W.
1883. PERIGAL, ARTHUR, M.D., New Barnet, Herts.
1876. PHILLIPS, CHARLES DOUGLAS FERGUSON, M.D., F.R.S.E., 10, Henrietta-street, Cavendish-square, W. c 3.
1873. PHILLIPS, GEORGE RICHARD TURNER, M.R.C.S., 24, Palace-court, Bayswater-hill, W. c 2.
1885. PHILLIPS, JOHN, M.D., 71, Grosvenor-street, W.
1883. PHILLIPS, SIDNEY PHILIP, M.D. Lond., 62, Upper Berkeley-street, Portman-square, W.
1878. PHILLIPS, SUTHERLAND REES, M.D., St. Ann's Heath, Virginia Water, Berks.
1883. PICK, THOMAS PICKERING, F.R.C.S., 18, Portman-street, W. c 2.
1884. PIESSE, C. H., M.R.C.S., 2, New Bond-street, W.
1883. PITTS, BERNARD, F.R.C.S., 109, Harley-street, Cavendish-square. c 5, s 2.
1890. POPE, HARRY CAMPBELL, M.D. Lond., 280, Goldhawk-road, Shepherd's Bush, W.



1873. PORT, HEINRICH, M.D., 48, Finsbury-square, E.C. *Hon. Sec. Foreign Correspondence.*
1850. \*POTTS, WILLIAM, F.R.C.S., 2, Albert-terrace, Regent's Park, N.W. c 3.
1871. POWELL, RICHARD DOUGLAS, M.D., 62, Wimpole-street, W. p, vp, c 5, o.
1891. POWELL, WILLIAM WYNDHAM, M.R.C.S., 34, Cliveden-place, Eaton-square, S.W.
1891. PRESTON, THEODORE JULIAN, M.R.C.S., Staff Surgeon, Royal Navy, 7, Caroline-place, Mecklenburgh-square, W.C.
1891. \*PRICKETT, MARMADUKE, M.D., 12, Devonport-street, Gloucester-square, W.
1885. PRINGLE, JOHN JAMES, M.B., 23, Lower Seymour-street, W.
1889. PRITCHARD, OWEN, M.D., 37, Southwick-street, Hyde Park, W.
1873. PURCELL, FERDINAND ALBERT, M.D., 7, Manchester-square, W.
1870. QUAIN, Sir RICHARD, Bart., M.D., F.R.S., 67, Harley-street, W. vp, c 3.
1883. RALFE, CHARLES HENRY, M.D., 26, Queen Anne-street, W.
1892. RAMSAY, JAMES, M.D., High Peter-gate, York.
1861. RAMSKILL, JABEZ SPENCE, M.D., 5, St. Helen's-place, E.C.
1881. RANKING, JOHN EBENEZER, M.D., Hanover House, Tunbridge Wells.
1859. \*RAYNER, JOHN, M.R.C.P. Edin., Swaledale House, Highbury-quadrant, N.
1890. RAYNER, WILLIAM, M.R.C.S., 4, Dorset-square, N.W.
1850. \*READ, REGINALD, F.R.C.P. Edin., 15 Windsor-road, Denmark-hill, S.E.
1879. REEVES, HENRY ALBERT, F.R.C.S. Edin., 7, Grosvenor-street, W.
1890. REID, JOHN, M.B., Clanmurray, Dromore, co. Down.
1882. REID, THOMAS WHITEHEAD, F.R.C.P. Edin., 34, St. George's-place, Canterbury.
1887. REMFRY, LEONARD, M.D., 60, Great Cumberland-place, Hyde Park, W.
1872. REYNOLDS, JOHN RUSSELL, M.D., F.R.S., 38, Grosvenor-street, W. c 3.
1872. RICHARDS, JOSEPH PEEKE, M.R.C.S., 6, Freeland-road, Ealing, W. c 3.
1850. \*RICHARDSON, Sir BENJAMIN WARD, M.D., LL.D., F.R.S., 25, Manchester-square, W. p, vp, LL, c 5, o, FM 1854.
1830. \*ROBERTS, HENRY PRATT, F.R.C.S., 31, Great Coram-street, W.C. vp 2, s 9, c 10, FM 1844, SM.
1891. ROBB, JAMES TAYLOR, M.D., 33, Lowndes-street, Belgrave-square, W.
1868. \*ROBERTS, DAVID LLOYD, M.D., F.R.S.E., 11, St. John's-street, Manchester.
1857. ROBERTS, DAVID WATKIN, M.D., 56, Manchester-street, W.
1885. ROBERTS, EDWARD COLERIDGE, M.R.C.S., Southgate, N.
1874. ROBERTS, FREDERICK THOMAS, M.D., 102, Harley-street, W. *Vice-President.*
1889. ROBERTS, Sir WILLIAM, M.D., F.R.S., 8, Manchester-square, W. c.

1873. ROBERTSON, WILLIAM HENRY, M.D., J.P., 6, The Square, Buxton, Derbyshire.
1884. ROBINSON, ARTHUR HENRY, M.D., The Infirmary, Bancroft-road, N.E.
1847. \*ROGERS, WILLIAM RICHARD, M.D., 56, Berners-street, W. vp, c 6.
1890. ROOT, ARTHUR GUERNSEY, M.D., Albany, New York, U.S.A.
1886. ROSE, ROBERT DUNCAN, F.R.C.S., St. Leonard's-place, York.
1874. ROSE, WILLIAM, F.R.C.S., 17, Harley-street. c 2, LL, vp 2.
1883. \*ROSS, DANIEL MCCLURE, F.R.C.S., 76, Upper Berkeley-street, W.
1888. \*ROTH, BERNARD, F.R.C.S., J.P., 29, Queen Anne-street, W.
1876. ROUTH, ALFRED CURTIS, 33, Marina, St. Leonards-on-Sea.
1881. ROUTH, AMAND, M.D., 14A, Manchester-square, W. c 2.
1848. \*ROUTH, CHARLES HENRY FELIX, M.D., 52, Montagu-square, W. p, vp 2, o, LL, s 4, c 6, SM. *Trustee.*
1891. RUFFER, MARC ARMAND, M.D., 5, York-terrace, Regent's Park, N.W.
1887. RUSHWORTH, FRANK, M.D., "Langdale," Goldhurst-terrace, South Hampstead, N.W.
1889. RUSSELL, JAMES SAMUEL RISIEN, M.B., 4, Queen Anne-street, W.
1886. RUTHERFOORD, HENRY TROTTER, M.D.
1879. RYLEY, JAMES BERESFORD, M.D., 53A, Welbeck-street, W.
1887. SAINSBURY, HARRINGTON, M.D., 63, Welbeck-street, W.
1884. SALTER, THOMAS KNIGHT, M.R.C.S.
1863. \*SANSOM, ARTHUR ERNEST, M.D., 84, Harley-street, W. vp, s 2, c 5, SM, §, LL, O.
1886. SAVAGE, GEORGE HENRY, M.D., 3, Henrietta-street, W. c.
1886. SAVILL, THOMAS DIXON, M.D., 12, Upper Berkeley-street, W.
1873. SEDGWICK, JAMES, M.D., Boroughbridge, Yorkshire.
1868. SEDGWICK, LEONARD WILLIAM, M.D., 2, Gloucester-terrace, Hyde Park, W. vp 2, c 4, § 3.
1883. SEMON, FELIX, M.D., 39, Wimpole-street, W. §, c.
1887. SERVAIS, LEOPOLD, M.D., Antwerp, Belgium.
1876. SEWELL, CHARLES BRODIE, M.D., 21, Cavendish-square W. c.
1889. SHAW, GEORGE, M.B., 1, The Drive, West Brighton.
1884. SHAW, JOHN, M.D., Burlington House, Willoughby-road, Hampstead, N.W.
1886. SHEILD, ARTHUR MARMADUKE, F.R.C.S., 20, Stratford-place, Oxford-street, W. c, s 2. *Councillor.*
1890. SHEPPARD, WILLIAM JOHN, M.D., Laurel House, Putney, S.W.
1871. SHETTLÉ, RICHARD CHARLES, M.D., 73, London-street, Reading.
1881. SHIPTON, ARTHUR, F.R.C.S. Edin., Buxton, Derbyshire.
1878. SHIPTON, WILLIAM PARKER, M.R.C.S., J.P., Buxton, Derbyshire.
1885. SHOEMAKER, JOHN V., M.D., 1031, Walnut-street, Philadelphia, U.S.A.
1890. SILK, JOHN FREDERICK WILLIAM, M.D., 29, Weymouth-street, W.
1890. SIMON, ROBERT M., M.D., 27, Newhall-street, Birmingham.
1884. SIMPSON, JAMES HERBERT, M.D., The Crescent, Rugby, Warwickshire.

1884. SINCLAIR, JOHN, M.R.C.P., General Post Office, St. Martin's-le-Grand, E.C.
1891. SISLEY, RICHARD, M.D., 11, York-street, Portman-square, W.
1883. \*SKERRITT, EDWARD MARKHAM, M.D., Richmond Hill, Clifton.
1886. SLATER, CHARLES, M.B., 16, Northwick-terrace, St. John's Wood, N.W.
1862. SLIGHT, GEORGE, M.D., 37, Western-street, King's-road, Brighton. c 2.
1889. SMALE, MORTON, M.R.C.S., 22A, Cavendish-square, W.
1845. \*SMILES, WILLIAM, M.D., St. Martha's Lodge, Guildford. vp 2, s 4, c 9, SM.
1887. SMITH, FREDERICK JOHN, M.D., 4, Christopher-street, Finsbury-square, E.C.
1848. \*SMITH, HENRY, F.R.C.S., 7, Wimpole-street, W. p, vp, LL, o, c 3.
1882. SMITH, HERBERT URMSON, Oudtshorne, Cape of Good Hope, South Africa.
1873. SMITH, HEYWOOD, M.D., 18, Harley-street, W. c 3.
1880. SMITH, NOBLE, F.R.C.S. Edin., 24, Queen Anne-street, W.
1891. SMITH, SOLOMON CHARLES, M.D., 4, Portman-mansions, Baker-street, W.
1877. SMITH, SYDNEY LLOYD, M.R.C.S., 25, Argyle-square, King's Cross, W.C.
1882. SMITH, THOMAS FREDERICK HUGH, F.R.C.S., Farningham, Kent.
1873. \*SMITH, THOMAS GILBART, M.D., 68, Harley-street, W. vp 2, s 2, SM, c 4. *Trustee.*
1872. SMITH, WALTER, M.R.C.P. Edin., 2, Stanhope-terrace, Gloucester-gate, Regent's Park, N.W.
1874. SMYTH, WILLIAM WOODS, Maidstone.
1888. SPENCER, JOHN, Lyons-terrace, Hetton, Durham.
1869. SPENDER, JOHN KENT, M.D., 17, Circus, Bath. FM 1874.
1887. SPICER, SCANES, M.D., 28, Welbeck-street, W.
1883. SPITTA, EDMUND JOHNSON, M.R.C.S., Ivy House, Clapham Common, S.W.
1864. SQUIRE, ALEXANDER JOHN BALMANNO, M.B., 24, Weymouth-street, Portland-place.
1881. STARTIN, JAMES, M.R.C.S., 15, Harley-street, W.
1892. STAVELEY, WILLIAM H. C., F.R.C.S., 13, South Eaton-place, S.W.
1884. STEPHENS, WILLIAM JOHN, 9, Old Steyne, Brighton.
1892. STEWART, HASTINGS, M.R.C.S., 13A, Charles-street, St. James's, S.W.
1882. STEWART, JAMES, F.R.C.P. Edin., Dunmurry, Sneyd-park, near Clifton.
1891. STEWART, WILLIAM EDWARD, F.R.C.S. Edin., 16, Harley-street, W.
1883. STEWART, WILLIAM ROBERT HENRY, F.R.C.S. Edin., 42, Devonshire-street, Portland-place, W.
1884. STIVEN, EDWARD WINNAN FLEMING, M.D., Lincoln House, Harrow, Middlesex.
1885. STIVENS, B. H. LYNE, M.D., 11, Kensington Gardens-square, W.
1848. \*STOCKER, JOHN SHERWOOD, M.D., 2, Montagu-square, W. c 10, s 2.
1884. STOKER, GEORGE, 14, Hertford-street, Mayfair, W.



1892. STONHAM, CHARLES, F.R.C.S., 4, Harley-street, W.  
 1877. STOWERS, JAMES HERBERT, M.D., 41, Finsbury-square, E.C.  
 1873. STRANGE, WILLIAM HEATH, M.D., 5, Grosvenor-street, W. c 3.  
 1881. STURGE, WILLIAM ALLEN, M.D., Maison Malausséna, Boulevard  
 Dubouchage 29, Nice. SM.  
 1889. SUMPTER, WALTER JOHN ERNELY, M.R.C.S., Sheringham, Norfolk.  
 1892. SUNDERLAND, SEPTIMUS, M.D., 35, Bruton-street, W.  
 1876. \*SUTHERLAND, HENRY, M.D., 6, Richmond-terrace, Whitehall, S.W.  
 1892. SUTTON, J. BLAND, F.R.C.S., 48, Queen Anne-street, W.  
 1892. SWIFT, WILLIAM JOHN CROPLEY, M.R.C.S., 4, Gordon-square, W.C.  
 1885. \*SYERS, HENRY WALTER, M.D. Camb., 3, Devonshire-street, Port-  
 land-place, W.  
 1884. SYMONDS, HORATIO PERCY, F.R.C.S., 35, Beaumont-street, Oxford.  
  
 1864. TAIT, EDWARD WILMSHURST, 48, Highbury-park, N.  
 1879. \*TAIT, LAWSON, F.R.C.S., 7, The Crescent, Birmingham.  
 1875. TAMPLIN, CHARLES HARRIS, "Lindfield," Crescent-road, Ramsgate.  
 1882. TAYLOR, SEYMOUR, M.D., 16, Seymour-street, Portman-square, W. c 2.  
 1859. THOMPSON, EDMUND SYMES, M.D., 33, Cavendish-square, W. VP, O,  
 S 3, C 3, SM.  
 1855. THOMPSON, Sir HENRY, F.R.C.S., 35, Wimpole-street, W. VP., LL, C 4.  
 1873. THOMSON, JOHN ROBERTS, M.D., Monkchester, Bournemouth, Hants.  
 1884. THOMSON, WILLIAM SINCLAIR, M.D., 1, Palace-court, Notting Hill  
 Gate, W.  
 1892. THORNTON, GEORGE, M.D., St. Marylebone Infirmary, Notting Hill, W.  
 1876. THORNTON, JOHN KNOWSLEY, M.C., 49, Montagu-square, W. P, VP, C 3.  
 1867. THOROWGOOD, JOHN CHARLES, M.D., 61, Welbeck-street, W. LL,  
 S 2, SM, C 3.  
 1856. THUDICHUM, JOHN LOUIS WILLIAM, M.D., 11, Pembroke-gardens,  
 Kensington, W. VP, LL, O, C.  
 1884. THURSFIELD, THOMAS WILLIAM, M.D., J.P., Selwood, Beauchamp-  
 square, Leamington.  
 1876. TIBBITS, HERBERT, M.D., 68, Wimpole-street, W.  
 1867. TIMMS, GODWIN WILLIAM, M.D., 9, Wimpole-street, W.  
 1865. TRAVERS, WILLIAM, M.D., 2, Phillimore-gardens, Kensington, W. c.  
 1884. \*TREVES, FREDERICK, F.R.C.S., 6, Wimpole-street, W. c 2. *Vice-  
 President, Lettsomian Lecturer.*  
 1882. TUKE, CHARLES MOLESWORTH, Chiswick House, Chiswick.  
 1886. TUKE, THOMAS SEYMOUR, M.B. Oxon., Chiswick House, Chiswick.  
 1884. TURNER, GEORGE R., F.R.C.S., 49, Green-street, Grosvenor-square, W.  
 C. *Councillor.*  
 1890. TWEED, EDWARD REGINALD, M.D., 55, Upper Brook-street, W.  
 1883. TWEEDY, JOHN, F.R.C.S., 100, Harley-street, W.  
 1891. TYSON, WILLIAM JOSEPH, M.D. Durham, 10, Langhorne-gardens,  
 Folkestone.

# XLIII

1887. \*UNDERWOOD, EDWARD T., M.D., Fort Bombay, India.
1883. VENNING, EDGCOMBE, F.R.C.S., 30, Cadogan-place, S.W.
1874. VERLEY, REGINALD LOUIS, F.R.C.P. Edin., 28B, Devonshire-street, Portland-place, W.
1892. WAGGETT, ERNEST BLECHYNDEN, M.B., 66, Park-street, Grosvenor-square, W.
1850. \*WAGGETT, JOHN, M.D., Perivale, Bournemouth; and Union Club, S.W.
1889. WAKEFIELD, THOMAS, M.B., 21, Beaumont-street, Marylebone, W.
1884. WAKLEY, THOMAS, 5, Queen's-gate, S.W.
1850. \*WAKLEY, THOMAS HENRY, F.R.C.S., 5, Queen's-gate, S.W.
1880. WALSHAM, WILLIAM JOHNSON, F.R.C.S., 27, Weymouth-street, W. c.
1881. WARNER, FRANCIS, M.D., 5, Prince of Wales-terrace, W.
1883. WATERHOUSE, WILLIAM DAKIN, LL.D., 18, Woodchurch-road, West Hampstead, N.W.
1872. WATERS, JOHN, M.R.C.S., 41, Bloomsbury-square, W.C.
1891. WATSON, W. SPENCER, F.R.C.S., 7, Henrietta-street, Cavendish-square, W.
1889. WAUGH, HENRY DUNN, M.D., 6, Sumner-place, Onslow-square, S.W.
1884. WEBB, F. ERNEST, M.R.C.S., 113, Maida-vale, W.
1889. WEBER, HERMANN, M.D., 10, Grosvenor-street, W.
1887. WEBSTER, HENRY WILLIAM, M.D., St. George's Infirmary, Fulham-road, S.W.
1838. \*WELLS, JOHN ROBINSON, F.R.C.S., 4, Pierrepont-road, Acton, W. c 2.
1884. WEST, SAMUEL, M.D., 15, Wimpole-street, W. s 2, c 3, CFC. *Councillor*.
1889. WETHERED, FRANK JOSEPH, M.D., 34, Queen Anne-street, W.
1892. WHEATON, SAMUEL WALTON, M.D., 52, The Chase, Clapham Common, S.W.
1882. WHIPHAM, THOMAS T., M.D., 11, Grosvenor-street, W. sm, c.
1884. WHISTLER, WILLIAM MACNEILL, M.D., 17, Wimpole-street, W.
1889. WHITE, E. F., F.R.C.S., Westlands, 280, Upper Richmond-road, Putney, S.W.
1868. WHITE, JOSEPH, F.R.C.S. Edin., Oxford-street, Nottingham.
1880. \*WHITE, WILLIAM HENRY, M.D., 43, Weymouth-street, W. c 3.
1885. WHITE-COOPER, GEORGE OWEN, M.B., 5, Cranley-gardens, S.W.
1883. WHITEHEAD, WALTER, F.R.C.S. Edin., F.R.S.E., 499, Oxford-road, Manchester. *Councillor*.
1885. WHITLA, WILLIAM, M.D., 8, College-square North, Belfast, Ireland.
1877. WHITMORE, WILLIAM TICKLE, 7, Arlington-street, Piccadilly, W.
1872. WILLIAMS, CHARLES THEODORE, M.D., 2, Upper Brook-street, Grosvenor-square, W. p, vp 2, ll, s 2, sm, o, l 3, c 9.
1876. WILLIAMS, HENRY WILLIAM, M.D., Hillside, Guilsborough, Northampton.
1883. WILLIAMS, JOHN, M.D., 63, Brook-street. c 3.

1883. WILLIS, ARTHUR KEITH, M.A. Oxon., Gascony House, West End-lane, N.W.
1881. WILLS, CALEB SHERA, C.B., Brigade Surgeon, Lunecliffe, Lancaster.
1873. WILLS, THOMAS MUNNS, F.R.C.S.I., J.P., 44, Merton-road, Bootle, Liverpool.
1893. WILLS, WILLIAM ALFRED, M.D., 23, Lower Seymour-street, W.
1892. WILSON, CLAUDE, M.D., Belmont, Tunbridge Wells.
1884. WINSLOW, H. FORBES, M.D., 14, York-place, Portman-square, W.
1873. WINSLOW, LYTLETON STEWART FORBES, M.B., D.C.L., 33, Devonshire-street, W. c.
1876. WOAKES, EDWARD, M.D., 78, Harley-street, W.
1882. WOLFENDEN, RICHARD NORRIS, M.D., 35, Harley-street, W.
1886. WOOD, T. OUTTERSON, M.D., 40, Margaret-street, Cavendish-square, W. c 3.
1873. WOODHOUSE, ROBERT HALL, M.R.C.S., 1, Hanover-square, W.
1889. WOOLFSON DE, LOUIS E. G., 26, St. John's-hill, Shrewsbury.
1891. WOOLLETT, CHARLES JEROME, F.R.C.S., 35, Telfourd-avenue, Streat-ham-hill, S.W.
1886. WORDSWORTH, WILLIAM JOHN, 4, Tisbury-road, West Brighton.
1884. WYMAN, WILLIAM SANDERSON, M.D., Red Brae, Putney-hill, S.W. c 2.
1891. YARR, MICHAEL THOMAS, Surgeon-Captain, Medical Staff, Junior Army and Navy Club, St. James's-street, S.W.
1884. YEO, I. BURNEY, M.D., 44, Hertford-street, Mayfair, W.
1884. YOUNGER, EDWARD GEORGE, M.D., 19, Mecklenburgh-square, W.C.

---

## NON-SUBSCRIBING FELLOWS.

---

1868. BATEMAN, Sir FREDERIC, M.D., J.P., Upper-street, Giles-street, Norwich.
1868. BEATTY, THOMAS CARLYLE, Seaham Harbour, Durham.
1872. BELL, JOHN HOUGHAM, M.D., Leconfield, Bonchurch, Isle of Wight.
1868. BUCKLE, FLEETWOOD, M.D., Staff Surgeon R.N., Southsea.
1868. CHILD, EDWIN, New Malden, Surrey.
1870. CLOUSTON, THOMAS SMITH, M.D., Royal Asylum, Morningside, Edinburgh. fm 1870.
1868. FLETCHER, THOMAS BELL ELCOCK, M.D., J.P., 8, Clarendon-crescent, Leamington.
1868. FOLKER, WILLIAM HENRY, F.R.C.S., Hanley, Staffordshire.



# XLV

1869. FOSTER, Sir WALTER B., M.D., M.P., 14, Temple-row, Birmingham.
1868. FOX, CHARLES HENRY, M.D., Brislington House, near Bristol.
1868. FOX, JOHN MAKINSON, The Grove, Lymm, Cheshire.
1868. GAINE, CHARLES, 30, Gay-street, Bath.
1871. GLYNN, THOMAS ROBINSON, M.D., 62, Rodney-street, Liverpool.
1872. HARRIS, HENRY, LL.D., M.D., Redruth, Cornwall.
1868. KIRKMAN, WILLIAM PHILLIPS, M.D., Elphinstone-road, Hastings.
1868. KNAGGS, SAMUEL, Ebor Mount, Huddersfield.
1869. LEES, CHARLES ALEXANDER, M.D., Fleet Surgeon R.N.
1869. LIPSCOMB, JOHN THOMAS NICHOLSON, M.D., St. Albans, Herts.
1869. LUNN, WILLIAM JOSEPH, M.D., Hull.
1859. MARSHALL, JAMES, M.D., 6, Rubislaw-place, Aberdeen.
1869. MATHEWS, ROBERT, Bickley, Kent.
1871. MAURICE, OLIVER CALLEY, 75, London-street, Reading.
1862. MAYBURY, AUGUSTUS KINGSTON, M.D., Holly Lodge, Richmond,  
Surrey.
1868. MCINTYRE, JOHN, M.D., LL.D., Odiham, Hants.
1868. NEVINS, JOHN BIRKBECK, M.D., 3, Abercromby-square, Liverpool.
1871. OGLE, WILLIAM, M.D., The Elms, Derby.
1869. PEMBERTON, OLIVER, F.R.C.S., J.P., 65, Temple-row, Birmingham.
1869. PHILIPSON, GEORGE HARE, D.C.L., M.D., J.P., 7, Eldon-square,  
Newcastle-on-Tyne.
1869. PRICE, WILLIAM PRESTON, M.D., 1, Ethelbert-crescent, Margate.
1869. PRIOR, CHARLES EDWARD, M.D., St. Peter's, Bedford.
1869. ROBERTS, BRANSBY, M.D., Badlesmere House, Eastbourne.
1871. SLOMAN, SAMUEL GEORGE, Farnham.
1869. STEAR, HENRY, Saffron Walden, Essex.
1869. TAYLOR, CHARLES BELL, M.D., 9, Park-row, Nottingham.
1869. WEBSTER, FREDERICK RICHARD, St. Albans, Herts.
1868. WIBLIN, JOHN, F.R.C.S., Clewer, Windsor.

\*.\* As it is very desirable that the List of Members should be kept as accurately as possible, Fellows are requested to send notice of any corrections that may be necessary to the Secretaries or to the Registrar.

---

## GENERAL MEETING.

---

*March 6th, 1893.*

JONATHAN HUTCHINSON, F.R.C.S., F.R.S., President, in the Chair.

*Annual Report of the Council presented at the General Meeting of the Society, March 6th, 1893.*

ACCORDING to custom, the Council begs to submit to the Fellows its Annual Report for the past session.

The meetings have been, as a rule, well attended, and papers of interest and importance have been brought forward.

The Clinical Evenings have been much appreciated, and the Secretaries take this opportunity of soliciting the co-operation of the Fellows in assisting them to find cases of interest, as on several occasions some difficulty was experienced in providing sufficient clinical material.

The Annual Oration by Sir James Crichton Browne, F.R.S., was delivered before a crowded audience, and the Lettsomian Lectures by Dr. John S. Bristowe, F.R.S., were numerously attended and excited much interest.

The Anniversary Dinner at the Hôtel Metropole, with Dr. Douglas Powell, President, in the Chair, was a social success, no less than 163 Fellows being present. The Annual Conversazione was well attended and generally appreciated.

The total number of Fellows on the roll is 764, of whom 590 are Subscribing Fellows. The Society is to be congratulated on the large number (39) of new Fellows who have joined it in the past session. Two former Fellows have been readmitted, and there have been ten resignations. The Council has to deplore the loss by death of 15 Fellows, leaving a net increase of 16 Fellows during the whole session.

The Council must make special reference to the decease of Sir Richard Owen. His death deprives the Society of one of its most illustrious Honorary Fellows, whose life-work is too well known to need recall here. Sir William Aitken, whose name also occurs among the list of deceased Fellows, was well known to the profession through his treatise on medicine and his valuable work in connection with the Army Medical Service.

The special advantages of the Medical Society of London are well known, and the papers published in the annual volume of Transactions cover the whole range of medicine and surgery. The Council hopes that the Fellows will endeavour to induce others to join the Society, and so fill the vacancies occasioned by death or resignation.

Vol. XV of the Transactions was presented at the first meeting in October last. It includes the Oration on "Sex in Education," by Sir James Crichton Browne, and the Lettsomian Lectures on "The Surgery of Trigeminal Neuralgia," by Professor William Rose. Among other papers of great interest in this volume may be mentioned "Observations on the Cure of Ascites due to Hepatic Disease," by Dr. Bristowe, and "Further Observations in Hepatic Surgery," by our former President, Mr. Knowsley Thornton.

The first award under the new scheme for the administration of the Fothergillian Trust was made in March. A gold medal of the value of 20 guineas, together with the full honorary premium of 40 guineas, has been granted to Dr. W. R. Gowers, F.R.S., for his work in practical medicine, especially with reference to his important treatise on 'Diseases of the Nervous System.'

The financial condition of the Society is satisfactory, as will be seen by the balance-sheet appended hereto. In the last Report reference was made to extensive building operations carried out immediately adjacent to the Medical Society's premises. Without giving details of the various arrangements which have been entered into, it will suffice to say that, by the yielding of a small portion of frontage in Chandos Street, the Society has gained material advantage in the building for it of additional premises, and an undertaking on the part of the builder to carry out by March, 1894, certain important alterations in the present ground-floor, which will include a new cloak room and lavatories.

In addition to the above, the Society received a money payment of £500, whereby the Treasurer and Council were enabled to pay off six bonds, reducing the debenture debt of the Society to £2,100, and effecting a proportionate decrease in the annual interest on the bonds. This satisfactory termination to lengthy and complex negotiations was not arrived at without much trouble; and the best thanks of the Society are due to the House and Finance Committee, and especially to its Chairman, Mr. Goodsall, whose untiring exertions on behalf of the Society entitle him to the hearty thanks of every Fellow.

The increased work and the disturbance associated with building operations have interfered with the progress of several important matters which the Council hoped to have carried to completion. Foremost among these is a complete revision of the laws of the Society, which are, to a certain extent, antiquated and confused. A beginning has already been made, and the Council hopes to complete the revision during the next session.

The Council wishes to express its appreciation of the services of the Registrar, Mr. Hall, during the past session. He has shown the greatest energy and zeal in undertaking greatly increased duties amid considerable difficulties, in consideration of which, and on the recommendation of the Treasurer and House and Finance Committee, his salary has been raised to £100 per annum.

In conclusion, the Council has to congratulate the Society in retaining for another year the services of its Treasurer, Mr. Arthur E. Durham, whose excellent management of the finances of the Society is well known and recognized and entitles him to hearty commendation.

The best thanks of the Society are also due to the Hon. Librarian, Dr. Allchin, for his unremitting attention—under the aforementioned disturbing circumstances—to the requirements of the Library, and the Council is fortunate in being able again to retain his services.



*Report of the Honorary Librarian.*

I regret that the Report which I have the honour to present on the work of the Library during the past year is scarcely so satisfactory as that which I have submitted on former occasions, owing to the very serious interruption and inconvenience brought about by building operations. It is sincerely to be hoped that, when the present alterations are completed, opportunity will offer for completing the laborious and lengthy work which still remains to be accomplished before the Library can be regarded as being in that state which the Fellows of the Society have a right to expect.

During the past year 259 volumes have been added to the Library, 50 of which were presented by authors or publishers and the remainder purchased from the Fothergillian Fund. A large number of magazines was also presented by Dr. Mitchell Bruce.

The card catalogue of English medical works of the 16th and 17th centuries is almost completed, and Mr. Hall has, with much labour, catalogued the greater bulk of the pamphlets.

Some progress has been made in the repairing and rebinding of the older works in the cases in the Meeting Room, towards the expense of which a small grant has been made from the funds of the Society, and a donation has been given by Sir Andrew Clark, a former President, and another is promised by Sir Richard Quain.

The Library Committee has met three times during the year.

My thanks are specially due to the Librarian, Mr. Hall, for most valuable assistance—rendered often at much personal inconvenience—for which the Society is much indebted to him.

W. H. ALLCHIN, M.D.,  
*Hon. Librarian.*

---

RECEIPTS.		EXPENDITURE.	
1892, Feb. 1 to 1893, Jan. 31	£ s. d.	1892, Feb. 1 to 1893, Jan. 31	£ s. d.
To Balance from last Account . . .	64 18 3	By Rent, Gas and Electric Light, Coals, Rates, Taxes, and Insurance . . .	480 15 8
" Life Composition . . .	7 17 6	" Repairs . . .	43 8 1
" Subscriptions (including Arrears) . . .	531 5 0	" Library Expenses . . .	28 5 5
" Entrance Fees . . .	33 1 6	" Salaries, Gratuities, and Collector's pound- age . . .	136 10 3
" Rents . . .	719 18 6	" Gratuity to the Widow of the late Regis- trar (final instalment) . . .	50 0 0
" Contributions for use of Rooms . . .	23 2 0	" Printing and Stationery . . .	25 18 3
" Sale of Transactions . . .	1 10 0	" Printing and issuing Transactions, Vol. XV . . .	170 18 4
" Cash received <i>re</i> Leasehold, Chandos- street . . .	500 0 0	" Postage . . .	11 5 5
		" Interest on Debenture Bonds . . .	101 10 1
		" Wages . . .	48 10 2
		" Miscellaneous Expenses, including Refresh- ments at Meetings, Band at Dinner, Chandlery, and Cleaning, &c. . .	47 19 3
		" Conversatione . . .	45 0 0
		" Surveyor's Charges . . .	9 9 0
		" Redemption of 6 Bonds . . .	600 0 0
		" Balance at Bankers . . .	82 2 10
	£1881 12 9		£1881 12 9

FOTHERGILLIAN FUND.

	£ s. d.		£ s. d.
To Balance in Bank from last account . . .	145 3 0	By Books purchased for Library . . .	31 3 10
" 1 year's Div. on £916 10s. 5d. $2\frac{3}{4}\%$ . . .	25 4 0	" Legal Expenses . . .	3 3 0
		" Balance at Bankers . . .	136 0 2
	£170 7 0		£170 7 0

(Signed) ARTHUR E. DURHAM, *Treasurer.*

February 16th, 1893. Audited, compared with the vouchers, and found correct,

(Signed) C. J. CULLINGWORTH, } *Auditors.*  
C. B. KEETLEY, }





TRANSACTIONS  
OF THE  
MEDICAL SOCIETY OF LONDON.  
120TH SESSION.

*October 17th, 1892.*

OPENING ADDRESS ON NAMES, DEFINITIONS, AND  
CLASSIFICATIONS.

By the President, JONATHAN HUTCHINSON, F.R.C.S., F.R.S., LL.D.

GENTLEMEN,—I propose, with your permission, to avail myself of your President's chair in order to make a few suggestions and criticisms in reference to a topic of great importance, and, at the same time, of much difficulty. I allude to the necessity, which is probably felt by most investigators, for a large reform in medical nomenclature, for better classification of disease, and, above all, for more precise definitions of the terms which we use. I can scarcely hope to escape the charge of temerity in venturing on such a theme—so wide in its bearings and so intricate in its details—but it will, I hope, be granted that the occasion is not wholly inappropriate, since I am addressing the members of the oldest medical society of our metropolis, and one which has always taken a foremost place in the advancement of clinical knowledge. It is precisely because great advance has been made in all departments of our knowledge as to the nature of disease that reforms in reference to the names which we employ have become needed. We have outgrown the nomenclature of our forefathers. It would be strange if it were not so. If the few and simple names which sufficed to express the diagnostic achievements of a century ago were sufficient for us now, the fact would imply disgrace. In every department of natural history new species have been discovered,

and new names devised for them. The number of the species of flowering plants which have now been discriminated and have received distinctive names amounts, I am told, to 100,000. For the multiform manifestations of the results of the innumerable influences which combine together to evoke disease in man, our names are probably not in the proportion of 1 per cent. I am aware that there exists an intense dislike to the introduction of new names, and more especially to all attempts at new classification. To some extent this prejudice is well based; yet, having regard to the future progress of clinical knowledge, I here make my most earnest protest against the present restriction of our nomenclature and the artificiality of our classifications. They constitute, I feel sure, hindrances of the very first moment. The surgeon who confines his attention to fractures and dislocations, aneurysm, hernia, and a few other cognate subjects, may easily get along with the present system. His subjects of investigation are, to a large extent, mechanical and almost wholly and strictly local. It is not a matter of any great difficulty to name, describe, and classify them. So, too, may the physician of the older school, who is content with his phthisis, bronchitis, and pneumonia, his albuminuria, diabetes, and gout. So, too, may the specialist make himself happy in the success of his iridectomies and his cataract extractions, and take very legitimate pleasure in the accuracy with which he can rectify by lenses defects in the configuration of the eyeball.

Let us not for one moment underrate the importance of these every-day achievements, or of that common knowledge of common things which is a *sine quâ non* to all of us. Our concern is rather to assert strongly that as soon as the specialist attempts to transcend his specialism, as soon as the physician or surgeon tries to get out of routine, and makes the attempt to understand not alone some, but all, the problems of pathogenesis which are brought before him, so soon will each and all find themselves cramped and hindered by our narrow and conventional systems of nomenclature and our fragmentary classifications. This is my assertion, and I make my appeal unhesitatingly to the experience of all clinical workers. I ask the neurologist whether such names as *tabes dorsalis* and *epilepsy* do not now each include a number of separate maladies which require separate grouping. I ask the physician whether he is content with the terms "diabetes" and "hysteria," while re-

specting dermatology I venture to offer my own opinion that there are at least twenty maladies classed under the name of lupus which require to be discriminated, and, putting all these aside, some five or six different types of "tuberculosis of the skin."

"SUBSTANTIVE DISEASE" AND "MORBID ENTITY."

I trust that no one will think it necessary to remind me that there is a great difference between the names and systems of the zoologist and those of the student of disease, or, at any rate, that he will not do it in a manner to suggest that he considers the remark original. Nothing can be more obvious than that names, definitions, and classifications are far more difficult in our department of natural observation than in any other. If "species," "genus," and the like in zoology have become in the light of modern discovery words which we must understand with some mental reserve, they yet retain to a large extent their old limited meanings, and are themselves capable of definition. In our special pursuits, however, such words have only an exceedingly restricted use. Few, indeed, are the forms of disease respecting which we can say, "This malady is *sui generis*." The clumsy but still indispensable expressions "substantive disease" and "morbid entity," employed as implying that the phenomena so denominated stand alone and complete in themselves, and are in relation with one single cause, are probably in use amongst us far oftener than sound knowledge of the facts would justify. The more we know, indeed, the less are we inclined to admit respecting any given disease that it is substantive and self-complete. For the most part in studying disease we have to deal with most complicated phenomena and with an almost inextricable interlacement of varied causation. Even in what appear to be the simplest of our problems, such, for instance, as the introduction of a specific poison into the system, we have to remember that one partner in the so-called disease is the living body of the patient, and that no two cases are exactly alike. But the fact that definitions and classifications are in the case of medical pursuits immeasurably more difficult than in most other branches of knowledge does not make them of any the less importance. An orderly arrangement of the materials before us is the first requisite to success in the pursuit of further discoveries; and just as an adequate vocabulary must of necessity precede the making of defi-



nitions, so must carefully studied definitions precede any attempt at classification which will not necessarily result in error.

#### DEFINITIONS AND DESCRIPTIONS.

Permit me here, gentlemen, *in limine*, to deprecate earnestly the suspicion of a wish to stereotype any given definitions or to fix the laws of classification. In a progressive science like ours any such aims would be absurd. All our efforts in these directions must of necessity be tentative and temporary. Not the less earnestly, however, do I make protest against that idleness of mind which is content to go on using words to which no definite meanings have ever been assigned, and to abstain from attempts at natural classification merely because they are difficult. A distinction must be drawn between a description and a definition. The former takes cognisance of the outward appearances of the disease described, and may be developed in much detail; the latter should be concise and, whilst seizing upon essential features, should exclude all that is extraneous. A good description should neglect nothing which may help to identification, whereas a definition may be very good, yet, from the fact that it concerns itself rather with abstract qualities, may give but little immediate aid towards the recognition of the thing defined. A description is a not difficult achievement to any good observer; a definition demands much thought as well as observation. A definition may often include many related objects, each of which would require a separate description. Thus, for example, it may be suggested that the definition of "keloid" should be "fibroid overgrowth of scar tissue"; but as being essentially of keloid nature we should have to describe for clinical purposes several affections, some of them subcutaneous, some extensively multiple, which offer at first sight little or no resemblance to the well-known glossy growths which so often develop in the scars of burns. A description of these latter, whilst it would offer by far the best aid to their diagnosis, would necessarily include features such as glossiness, brightness of colour, development of spurs, &c., which are wholly absent in some of the affections referred to. Although I would gladly excuse myself, I feel that I can scarcely with credit avoid the attempt to give some illustrations of what I have been trying to urge. We will, if you please, take the names of three common diseases and

try to define them, and at the same time to show how their definitions affect their position in any system of classification founded on natural affinities. Phagedæna, erysipelas, and diphtheria are words in common use, and yet I suspect that there is considerable difference of opinion as to how they should be defined, and as to where the diseases which they designate should be placed.

“PHAGEDÆNA.”

Respecting phagedæna, it may perhaps be said that it is not the name for a disease, but rather for a certain condition occasionally assumed by the inflammatory process. Permit me to assume, since time does not permit of our arguing the point, that between simple phagedæna, sloughing phagedæna, and hospital gangrene there is no difference of kind, but only of degree of severity—that hospital gangrene is simply phagedæna having become epidemic in a hospital containing patients affected with wounds. Permit me to assume, also, what is exceedingly probable, that the gangrenous affections which attack the mouths and genitals of young children, and which are known as noma and cancrum oris, are really of a closely similar nature, though of a more severe type, with the phagedæna which we so often encounter in syphilis. These data being granted, we have first to note that it is undoubted that cancrum oris, noma, and syphilitic phagedæna may begin quite independently of contagion. It is therefore not a specific malady in the sense of being dependent upon the implantation of a specific virus. Next, we must record that, although these inflammations may occur to persons seemingly in robust health, they are almost invariably those who have suffered from syphilis, or, in the case of noma and its congener, recently passed through a specific fever, measles, variola, whooping-cough. With somewhat more hesitation, another assertion must be made, that the use of mercury has often preceded not only the syphilitic forms, but also those seen in children. We come, then, to the definition of phagedæna as a locally destructive form of inflammation, which attacks sores and wounds, which varies much in severity in different cases, and which occurs for the most part in those who have recently suffered from some specific disease. To this must be added the important proposition that, although capable of spontaneous origin under the conditions mentioned, it is infective to adjacent parts, and may

become contagious to other patients with open wounds. We abstain from any description of a phagedænic wound, for the appearances may differ very widely in different cases. For classification purposes we have enough. Phagedæna is not to rank with the specific fevers, although it may become epidemic; it is a type form of specialised inflammation, occurring under certain known conditions of predisposition.

#### “ ERYSIPELAS.”

The term erysipelas is suggestive of controversy. For long there have been two schools of opinion: the one regarding it as a specific fever, and classing it with the exanthems; the other ranking it only as a specialised form of inflammation. The difference is very important. I am glad to observe that the philosophic Dr. Hirsch has ranged himself with the latter, and defines erysipelas as “an infective inflammation-disease of the skin or of one of the mucous membranes,” &c., and speaks of “the infective fever which accompanies the local process.” On the other hand, most recent English authorities prefer to regard it as an exanthem. The facts which oppose this claim appear to my mind to be definite and conclusive. When erysipelas attacks a wound, its development is always local, and the constitutional disturbance is in ratio with the extent of surface involved. It may develop without any appreciable incubation stage, and it may be cut short at any period by suitable local treatment. Its duration is not uniform, but may be indefinitely protracted. One attack does not prevent a second, but, on the contrary, predisposes to others. Thus, then, I come easily to the conclusion that erysipelas is a name for a form of inflammation presenting peculiar features, very infectious to the tissues (the lymph spaces) of the patient, easily spreading by contagion to the wounds of others, and of which one attack leaves proclivity to a second. We have to add to this, however—unless, in contradiction of all probability, we insist that medical and traumatic erysipelas are two quite different things—that it is capable of origin from exposure to cold and without either wound or contagion. Erysipelas is, it will be seen, placed according to this definition by the side of phagedæna, and just as it would be more correct to speak of phagedænic inflammation rather than of phagedæna, so it would be better to use the expression “erysipe-



latus inflammation" rather than "erysipelas," since the latter is too suggestive of a substantive disease which always runs the same course and always presents the same conditions.

### "DIPHTHERIA."

Let us now see how the case stands with diphtheria. It is interesting to note that this name has been substituted for what was not improbably a far more correct one—diphtheritis. What is there to hinder us from defining this affection as a contagious inflammation of mucous surfaces; and chiefly of the throat, frequently attended by the formation of pellicles infective by continuity in the patient, and easily spreading by contagion to others? It would surely be wrong to make the formation of the diphtheritic false membrane a part of the definition, for when the disease passes by contagion through a household there are always some cases which show very little membrane, and often a few in which there is none at all. We do violence to probable truth if we deny that these latter are diphtheritic cases, for they originate from contagion from those which undoubtedly are such. The medical officers of those metropolitan asylums which receive fever and diphtheria cases always in their reports count a certain number of cases sent to them as "errors in diagnosis," and of these a considerable number are "non-specific sore throats" sent in as "diphtheria." This is surely rather hard upon those who send them. It is to make the diagnosis of a disease depend, not upon its origin, but upon the degree of severity with which it develops certain peculiar phenomena which are common but not invariable. No man attending a number of patients with sore throats in a house with diphtheria can possibly tell which will develop extensive membranes and which may have none.

There are other important facts which ought to be taken into consideration in the definition of diphtheria. It is quite impossible to distinguish the sporadic cases of membranous croup from those of epidemic diphtheria, and admitting—as I think we must—that they are exactly the same, we are almost forced to conclude that a disease which becomes contagious may originate independently of contagion and in connection with exposure to cold and damp. In these points the facts as to diphtheria are exactly parallel with those as to erysipelas and phagedæna. All three occur sporadically and

in connection with individual proclivity rather than specific influences, yet all may become contagious, prevail as epidemics, and when they do so keep close to type.

I have ventured to attempt explanations of these three words, not with any object of giving model definitions, far less with any hope that what I have expressed will satisfy others, but rather to illustrate the importance of the matter and the lines upon which I think that we ought to proceed. I have freely admitted that the construction of a good definition is a most difficult achievement. Here, if anywhere, we need the light of many minds. I am sanguine, however, that if medical writers could be induced to preface their works with concise definitions of those names of disease which they employ, it would save them from much obscurity of diction and greatly advantage their readers.

I feel sure that I shall incur criticism for not admitting to a foremost place in the definition of such affections as diphtheritic and erysipelatous inflammation the parasitic microbes which have been demonstrated to be usually present. Whilst I fully admit the importance of the facts which have been established in respect to them, and think that it is highly probable that they do take a large share in the process of contagion, I am yet not convinced that they constitute the disease. It seems to me premature to adopt such a conclusion, and I think it safer for the present to trust to the statement of the broad clinical facts. It may be one thing that these parasites should be capable of conveying the disease, and another that it should invariably depend for its origin upon their implantation. I feel sure that truth will lose nothing in the end by a little hesitation in accepting all that seems to be true respecting these wonderful micro-organisms, and witnessing the enthusiasm of some of my friends, feel at times half inclined to exclaim with the poet:—

O! your parasite  
Is a most precious thing dropp'd from above,  
Not bred 'mongst clods and clodpolls here on earth.  
I muse the mystery was not made a science,  
It is so lib'rally profest! Almost  
All the wise world is little else in Nature  
But parasites or sub-parasites.

It is not only in reference to acute inflammations of the kind just mentioned that it is desirable to be cautious how we accept

the presence of a microbe as the final and sole explanation of origin. If we could do so safely it would certainly be a very great assistance to our definitions. The precise *rôle* of the bacillus in the disease leprosy is, I think, far from being as yet established, whilst it would certainly be very premature to define any one form of lupus as being the product of the bacillus of tubercle. If we did so, we should at once have on our hands a number of closely cognate maladies which could not be so explained, and which it would yet be impossible to put wholly aside. In the disease to which Hebra gave the name of rhinoscleroma the presence of a microbe has been demonstrated only in a few instances. Inasmuch as a number of others in which search has been fruitless present exactly the same clinical features, I think that the disease, which is well characterised, should take its definition independently of them. There are other maladies to which the same remark will apply.

#### THE INVENTION OF NEW NAMES.

It is mainly in reference to very rare maladies that the invention of new names is required. It is not to be expected that in the present day any common maladies will be discovered which our predecessors had wholly overlooked. It may be truthfully alleged, however, that the rare maladies, although rare, are for their illustrative value of extreme importance, and they ought as promptly as possible to become objects of general knowledge. It may further be asserted as regards common and well known diseases that there are a host of varieties which, were it practicable, are well worthy of distinctive recognition. The clinical investigator who sets himself resolutely to observe in detail and to classify with accuracy soon finds him at a loss for distinctive names for different groups of cases. To refuse him the privilege of a detailed nomenclature is as absurd as to insist that the ornithologist shall call all birds of prey either hawks or owls.

It becomes therefore of much importance to find out the most convenient method of giving names, what kind of names are the easiest to use and to remember and the least liable to suggest error. New names of some kind we must have; they are absolutely essential to progress. Allow me to mention, as illustrative of the difficulties which await us, a few of the new designations of



disease which have come into use during the last quarter of a century. Amongst those supposed to be from Greek roots and to be more or less descriptive names we have: Acromegaly, myx-œdema, mycosis fungoides, actinomycosis, xerostomia, xerodermia, and many others. Respecting them all we may object that they are intelligible only to Greek scholars, and not always to them; and to all others they are very unattractive, as, in addition to their being meaningless, they may possibly be difficult to remember and a little uncertain as to pronunciation and orthography. These, however, are minor drawbacks, the principal one being that it is impossible to condense into any Greek word, however composite, anything approaching to a correct description of a disease. Acromegaly, although one of the best, does not cover the whole ground, and suggests false limitations. Actinomycosis takes us no further than the assertion of the presence of a fungus which forms rays, although it must be admitted that so far it is very good. Mycosis fungoides is a name for a group of cases, many of which, at any rate in their early stages, do not fungate at all, and concerning which it is doubtful whether its origin is cryptogamic. Xerodermia, a name of easy etymology, has been used in connection with various adjectives in association with which it has been applied to maladies having no real relationship. One of these, xerodermia pigmentosa, or Kaposi's malady, has been found so difficult to find a classical name for that almost every author who has written on it has devised a new one.

#### THE USE OF THE DISCOVERER'S NAME.

Another class of names is that in which the name of the observer is affixed to the morbid phenomena observed. Bright's disease and Addison's disease supply us with examples of the very successful, though perhaps only temporary, use of proper names in this way. Amongst more recent ones we have Charcot's joints, Paget's disease or osteitis deformans, Paget's nipple, Menière's disease, Jacksonian epilepsy, and Raynaud's disease.

Most of these have probably been given in the first instance with the express design of doing honour to the individual whose name is made use of, but I suspect that those which have come into common use have attained their popularity simply because the

profession found them more convenient than any others. Such names, being meaningless so far as etymology goes, have a negative advantage over most others in that they do not mislead, and do not, either by limitation or excess, suggest anything which is false. The reader has but to make himself acquainted with the original paper in which the malady was described, and he may learn without risk of error to what cases the name ought to be applied. To this method of naming there is, however, the obvious objection that it cannot be indefinitely or even widely adopted. Excepting in reference to discoveries of foremost rank, the profession would certainly, and very properly, decline to use the name of any contemporary. Nor can such names be made use of early; we have to wait until the discovery has been ratified and acknowledged as really new by other observers before we can venture, without risk of grotesque failure, to affix the name of its discoverer to it. Nor at the best can it be considered that such names are quite consistent with the dignity of those to whom the honour is offered. They should, at any rate, be few and far between, and those whom we select for such kind of eminence should for the most part be those who have gone to their rest. Our profession is one which cannot be accused of lack of generous recognition of those who serve it, and it has plenty of other modes of expression at its command without naming diseases after living men. In saying this, however, I still wish to ask attention to the fact that such names have been found very convenient, and further, that amongst our zoological and botanical *confrères* proper names are so employed in the freest possible manner.

#### RULES FOR THE FORMATION OF NEW NAMES.

If, then, we revert to the proposition that names for new diseases or new varieties or combinations of symptoms should be selected solely with a view to the convenience of the profession, a few simple rules may, I think, be laid down. Such names should (1) be short and easily remembered; (2) they should be in the vernacular; (3) they should be available from the very beginning, and such as the observer himself may without indecorum apply; (4) they should have in themselves no meaning, the reader being left to find that out by reference to the writings of the observer who first named them.

## ON THE USE OF PATIENTS' PSEUDO-NAMES.

Permit me here to lighten my discourse by relating to you an anecdote. It was one which was told me by my friend Dr. Billings a few weeks ago. A non-medical member of a hospital committee, in replying to a toast, took occasion to observe that, although he applauded the profession in most things, he could not help feeling that in some things it was open to a charge of self-glorification. "For instance," he went on, "if a man has an interesting and new malady, you very probably get him photographed, you take him to a society and show him, possibly you even cut a bit out of him for the microscope, and then at the end of it all you name the disease after the doctor. Now it seems to me that it would be much more fair that it should have the patient's name." When we had again become grave I told Dr. Billings that the suggestion was no novelty, and that I had myself, though for other reasons, long been in the habit of naming diseases which were new to me after the patients who first displayed them. Thus I have often used the name "Penman's prurigo" in print, Penman being the pseudo-name of the lad whose portrait is given in the New Sydenham Society's 'Atlas.' *Let me say, however, that, for obvious reasons, I never used in public the real name.* I have also used the name of Philip Holmes to designate a series of cases of which a boy so named was my first example. The cases of multiple ulcers in the legs of young persons, which have recently claimed much attention both in London and Paris, had for many years had a special box in my cabinet assigned to them under the homely but convenient designation of Mrs. Bransford's legs. Quite recently I have learned, thanks to Dr. Colcott Fox, that Bazin had long ago named this malady "erythème induré des scrofuleux"—a name which is, I must think, open to the objection that it is somewhat misleading, since the malady has exceedingly little of the erythema character about it.

Many years ago I described a form of arthritis occurring in the subjects of inherited gout which disorganises the last joints of all the digits, and is very often attended by attacks of recurrent iritis, which also disorganises the eyes. My first patient was a girl named Mabey. I have published her case, with a portrait of her hands, and although I have done my best, I have never been



able to find any name for her malady so convenient as that which it has always had in my notebooks of Mabey's disease. The joint disease is a "last-joint-arthritis," or more suitably perhaps an acro-arthritis, and the eye disease is a relapsing iritis, but apart from the fact that there are other varieties both of acro-arthritis and of recurrent iritis very different from these, how shall we hope to find a Greek word which comprises a reference to the fact of the two existing together? If, however, it were permitted to call the disease after the patient, and let the expression "Mabey's disease" comprise the totality of the symptoms which she displayed, the reader would be compelled to refer to the published record of her case, or to some abstract of it, and would not be allowed to confuse himself by trying to obtain an idea of the malady from the etymology of its inadequate designation. It is a subject upon which I have thought much, and respecting which I have some little experience, and seriously I have no better suggestion to make to the systematic case collector than that he should at any rate temporarily designate what appear to him to be novelties by the names of the patients displaying them. Probably many of us already do this in private, and the question rather is should it be permitted in print. I think that it should, and indeed I see no alternative unless the art of clinical observation is to be allowed to lag. The present plan of trying to make a small number of names suffice for a vast variety of objects, and of slavishly grouping together, because our fathers did it, under one and the same name diseases which differ widely, is one which has encumbered us too long. Our young and zealous investigators must give their minds sea-room, and seek to see things for themselves and as they really are. Probably there is no better plan in aid of exactitude in clinical observation than to take "type cases," to follow them to their end, describe them in detail, and wherever possible to illustrate them by photography or by drawings. By the side of the type case place all others which really fit with it and none else.

Anyone who will adopt this plan will, I feel sure, be astonished to find how rapidly parallel cases of what he at first thought rare and exceptional accumulate. We have but to provide centres for crystallisation, and the deposits are thrown down apace. Nor will he, I feel sure, be disappointed in the results to his own mind in the development of powers of independence in observation. We are all of us too much prone unconsciously to diagnose (that

is, to name) first and to observe afterwards. We think that it is our own fault if our cases do not fit with what others have recorded, and with the orthodox nomenclature of the text-books. In a spirit of modesty, which is most creditable but at the same time most prejudicial, we undesignedly endeavour to make them fit. I remember once being put on my guard respecting the case-notes of a certain ward clerk, and being told that he was in the habit of taking down the patient's name and age with the recorded diagnosis, and then, in the quiet of his own room, filling in all the details from a book. Now, absurd as such a plan of case-taking may seem when thus nakedly stated, I fear it is one which we all of us to some extent adopt. We do not go to our books, for it is not necessary, but we have stored in our memories certain facts in association with certain words and names which run together so easily that it is very difficult to let the observation-faculty work uninfluenced by the shades and colours which they cast.

I have often thought that in such a subject as dermatology, for instance, it might be well for a time to throw aside all our names, and take good, well-illustrated case narratives, and sort them afresh. We should in this way escape the trammels which education has imposed, and we should see natural differences and resemblances which at present elude us. It is thus that it often happens that it is the partially ignorant man who makes a discovery which those better taught could not see. In him the observation-faculty is more free to exert itself. Perhaps there are few words to which we more habitually give a too limited meaning than to the term diagnosis. To make a diagnosis ought to mean to recognise so far as is possible the real nature of the disease in question. It is too generally used as if it meant to give a name.

#### CLASSIFICATION.

I have left myself but little time to deal with the last of my three topics. I will not detain you long upon it, for I know well that the very name of "classification" is a *bête noire* with many. It is supposed that the state of our knowledge does not as yet permit it. Allow me, however, to say a few short words about it. Let us remember the enormous gain to botanical science which resulted from the substitution of the natural system for that of Linnæus. Now, in our subjects we can scarcely be said to have

got even so far as the latter. Diseases of the skin have, perhaps, been classified with more elaboration than any other, yet not only has no really natural grouping been attempted, but the arbitrary classes and orders which have been arranged cannot, I think, be said to afford to the student anything approaching the amount of assistance which the scheme of Linnæus, artificial as it was, undoubtedly gave to the young botanist.

I suspect that most readers of our dermatological handbooks skip the classification altogether, and as a rule it will be noticed that the author himself rarely pays much attention to it. Yet, let me ask you seriously, Is it not possible to attempt something in the way of a natural classification of skin diseases which should be of the utmost value to the student? I take dermatology as an example, because I chance to be somewhat familiar with it, and because, with a self-distrust which will, I hope, not be severely blamed, I dare not enter upon matters proper to the physician's domain. What is possible in dermatology might, however, be attempted in general medicine. He who would classify with success must enter upon his task with courage. He must not be afraid of details: he must readily eschew all pedantry, and be determined that nothing shall induce him to put two things in the same family group unless he is sure of their relationship. He must be willing to forego the hope of completeness, and think but very lightly of finality.

Under these conditions, I certainly believe that a very useful classification of skin disorders, according to natural affinities, is both desirable and possible. We could not assign at once all maladies to their proper places. But this is not requisite. What is wanted is to effect such an orderly arrangement of classes and groups that, when once the real nature of a disease is recognised, there is a place ready for its reception. I would take a hint from botanists and name the orders after their most prominent and typical examples. Thus, the disease known as psoriasis having been carefully defined, I would place with it, in a psoriasis family, all forms of skin disease which conform to similar laws. So with acne, pemphigus, and many others. Perfectly natural groups might be formed of those caused by animal parasites and of those due to cryptogamic growths. Other groups might be constituted by those due to congenital peculiarity of tissue—Kaposi's malady and the like.



In every case real affinities—in other words, relationship by sameness of cause—should be the guide in the formation of our family groups.

Gentlemen, I must conclude, and in doing so I can only hope that the topics I have been bold to bring before you have not seemed to you unsuitable for this the first meeting of a new session of the Medical Society of London.

---

*October 24th, 1892.*

A CASE OF VOLVULUS OF THE SMALL INTESTINE,  
FOLLOWING A FALL, SUCCESSFULLY TREATED  
BY ABDOMINAL SECTION.

By GEORGE R. TURNER, F.R.C.S.

WILLIAM SMITH, aged 7, was brought to the Seamen's Hospital at 8.15 P.M., July 26th, 1891, with the history of having fallen from a height of 12 feet against the pole of a boat, and then into the mud of the river.

On admission he was much collapsed, and vomited several times—bilious matter. He soon became very restless, rolling himself about in bed; his legs were drawn up, and there was much abdominal pain complained of, principally in the right iliac fossa. There was considerable tenderness in this situation, but no abdominal distension, no external injury. There was some tenderness, but no flatus or fæces passed.

July 27th. Was sick once in the night. Bowels not open. Still pain and tenderness about abdomen. The boy was extremely restless, with an abdominal aspect, and at midday he vomited some bilious fluid, which, for the first time, had a strong fæcal odour. Dr. Spencer, to whom I am indebted for these notes, sent for me, and I first saw him some twenty-four hours after the accident. The boy had passed water twice since his admission, smoky and blood tinged. I decided to at once open the abdomen and ascertain the nature of the injury before general peritonitis set in. Chloroform was accordingly administered, and an incision

4 inches long made in the middle line between the umbilicus and pubes. There was no evidence of any rupture of the intestine, no flatus, fæces, blood, or fluid in the peritoneal cavity. The large intestine was natural; on examining the small intestine, an entanglement was felt just to the left of the middle line. The mass of entangled intestines was easily unravelled, and on passing the gut bit by bit through the fingers, about a foot of it was seen to be completely collapsed and flattened. The collapsed gut at either end passed abruptly into gut injected, but otherwise of normal appearance. Further examination of the small intestine disclosed another portion, some 2 feet in length, in a similar collapsed and flattened state. This second collapsed portion was separated from the smaller part first found by at least 3 or 4 feet of healthy intestine. No tear or rent was found in either mesentery or omentum, or band of any kind discovered. There was no adhesion of the intestine to the abdominal wall. The part of gut affected was the ileum; there was no peritonitis. The abdominal wound was sewn up in the usual way, and cyanide dressings with salicylic wool applied. Orders were given that beyond a little iced water and brandy the patient was to take nothing by the mouth, but to be fed as far as possible by nutrient suppositories. After the operation there was no further vomiting, and but for some restlessness on the 29th (which yielded to an hypodermic injection of morphia), his recovery was rapid and uninterrupted. He passed flatus on the second day after the operation, and his bowels acted naturally on the eighth day. The wound healed by first intention. There was no trace of blood in the water after the day of his admission; it was quite clear and natural, except for a temporary deposit of lithates, on July 30th.

The stools, when the bowels acted, had no trace of blood in them.

I confess, when I opened this boy's abdomen, I expected to find some rupture of the intestine, or injury to some other viscus (possibly the kidney). His collapse, vomiting, abdominal pain, and tenderness, all seemed to point to this. If I had expected to have found any form of intestinal obstruction I should, from the presence of tenesmus, have been on the look out for an acute intussusception rather than volvulus. The boy was in perfect health before his fall, and I think therefore it is only reasonable to attribute the intestinal entanglement to that as a cause. My idea

is that it produced a twist of one coil of intestine round another. I know that this especial form of volvulus is very rare. I have seen it, however, in a case of my own I published elsewhere, where there was also an intussusception of the small intestine produced by a polypus. Length of mesentery is said to be favourable to the formation of a volvulus. I noticed nothing remarkable in this respect; though the mesentery was certainly very freely movable, and examination of the whole length of the intestine easily and rapidly performed. This, and the successful issue of the case, I attribute to the early performance of abdominal section before intestinal distension and adhesion made rectification difficult, and general peritonitis the prognosis of any operation a gloomy one.

The pain referred to the right iliac region was probably due to some dragging on the lower ileum; this was the part of the small intestine which I first examined, and proceeded upwards from there towards the duodenum. I think that in cases such as this all the intestine should, if the patient's condition allow of it, be systematically examined, even if one cause of obstruction has been met with. In the case of my own I have already alluded to, there were no less than three distinct seats of obstruction (volvulus in two places and intussusception). A rent in the urinary bladder has before now been carefully sutured, and a similar one in the small gut overlooked.

I have ventured to bring this case before the Society, as I believe it to be, anyhow, one of very few cases of intestinal obstruction directly due to and immediately following a fall, and as an argument, if indeed any are now needed, for early abdominal exploration in certain cases of abdominal injury.

Mr. MARMADUKE SHEILD remarked that these cases were very rarely met with in practice. When grave intestinal symptoms appeared, so to speak, spontaneously, without obvious traumatism, there were still many who doubted the propriety of immediate operative interference; but, in a case such as that related, where the symptoms developed immediately after an injury, he thought that most surgeons would at once perform laparotomy.

Dr. FRANCIS HAWKINS said that the case recalled one which occurred fifteen years before, when he was pupil at a county hospital. The patient was a female between 35 and 40, of a stout build, who, when standing on a chair doing some housework, suddenly slipped and fell on the ground. When seen, the symptoms were those of acute intestinal obstruction, and the patient died about forty-eight hours after the accident. At the autopsy there was seen, just above the situation of the umbilicus, a



transverse twist, representing a figure of 8. When all tension had been removed by drawing the abdominal walls aside, the bowel immediately replaced itself. A consultation was held previous to the death of the patient to discuss the propriety of making an abdominal section; this, however, was not considered advisable.

Mr. F. BOWREMAN JESSETT congratulated Mr. Turner upon the success of his case, and quite agreed with him that the earlier abdominal section was performed in cases of intestinal obstruction the better. The incision, which, in acute cases, should be of sufficient length to afford ample room for inspection, was, in Mr. Jessett's opinion, always best made in the middle line, but in cases when the patient was in a very collapsed condition, he considered a small incision and drainage of the intestine was the best treatment, leaving the more radical operation to a future period, when the patient might be better able to support it. Mr. Jessett observed that Dr. Hermann, at the International Medical Congress held at Berlin, pointed out that he had performed certain experiments on dogs by which he thought he proved that the secretion of the mucous membrane and intestinal glands could not be distinguished from ordinary fæces. Mr. Jessett had, with the kind assistance of Dr. Wright, performed the following experiments to test this point. A dog being thoroughly anæsthetised, an incision was made into his abdomen and a loop of intestines withdrawn, 16 inches of which was detached from the remainder by dividing the intestine at each end. This loop was carefully washed out, by means of a funnel, with carbolic solution. The ends were then carefully invaginated and securely sewn up. The continuity of the shortened intestine was then restored by circular enterorrhaphy. The intestines were then all replaced into the abdomen and the wound in the parietes closed. No bad symptoms manifested themselves after the operation; the dog was killed twelve days afterwards, and the loop of intestine was found much distended and measuring a little over 3 inches in circumference. It contained about six ounces of greyish-green thickish fluid of a stercoraceous odour. The mucous membrane of the intestine was quite normal. The fæcal matter from the loop was found to consist entirely of a mass of leucocytes with a certain amount of detritus and a few fatty crystals. The result of these experiments Mr. Jessett considered of great practical importance in these cases of volvulus or constricted loops of intestine, in which the adhesions were such as to prevent the untwisting or releasing the constricted loop of intestine. The experiment pointed to the absolute necessity of establishing an anastomosis between the loop and an adjoining piece of the intestine so as to allow of the drainage, so to speak, of the loop, as well as restoring the continuity of the intestinal canal by lateral anastomosis above and below the constriction.

Mr. STAVELEY reported for Dr. Nichol, of Margate, the following case:—

On September 3rd, 1891, Dr. Nichol was called at midnight to see a previously healthy female child, aged 5 years, who was suffering from colicky pains in the abdomen. He elicited from the child that at 8 P.M. the same evening she had, whilst playing, struck the abdomen against an iron seat, but had not complained at the time and walked home, a distance of about a quarter of a mile. On examination, abdomen flaccid, no distension, percussion note normal. No external mark of injury. Some doubtful tenderness to left of umbilicus. Legs not drawn up. Pulse firm and vigorous, temperature normal, no signs of shock. She vomited a quantity of undigested food in Dr. Nichol's presence, and also passed urine. (Opium

and hot fomentations.) September 4th. Intermittent pains and vomiting of watery fluid had continued during the night. No action of bowels, but had passed urine freely. Condition of abdomen unaltered, but the localised tenderness was rather more marked. Temperature normal. Child looked wan and ill. 4 P.M. No pain or distension. Child collapsed, lips blanched, and extremities cold. A small ecchymosis had appeared just below right costal arch. 7.15. Died twenty-three hours after receipt of injury; no alteration in condition of abdomen. *Post-mortem*, eighteen hours after death. On opening abdomen there was a considerable quantity of blood-stained serous effusion. Several loops of small intestine deeply congested. About 30 inches from the pylorus the bowel was tightly obstructed by what appeared to be a band, but which, on further examination, proved to be a loop of intestine firmly twisted on its mesenteric axis. Dr. Nichol did not note any peculiarity in the anatomical arrangement of the mesentery.

Mr. HARRISON CRIPPS advocated a long incision in cases of exploratory laparotomy. Surgeons too often made a short incision, and were then much hampered in their inspection of the gut. The intestines should be allowed to crowd out of the wound and be received on a warm damp cloth. If there were much difficulty in returning them a puncture should be made, the contents evacuated, the incision closed, and the gut would then readily return.

The CHAIRMAN (Dr. Symes Thompson) pointed out that the case contributed by Dr. F. Hawkins and Mr. Staveley, taken in conjunction with Mr. Turner's case, demonstrated the fact that when abdominal section was performed sufficiently early, before gangrenous change or prostration had occurred, a fatal termination might unquestionably be prevented.

Mr. TURNER, in reply, said that the case related by Dr. F. Hawkins was of interest, apart from other considerations, as bearing on the fact that volvulus often wanted the resistance of the anterior abdominal parietes for its causation. This had already been shown by Melchiori's experiments. The case related by Mr. Staveley was peculiar, in that the volvulus affected the upper part of the small intestine. As a rule, it was the lower ileum that was implicated. He certainly would open the abdomen in such a case. Mr. Jessett's observations and experiments were interesting and suggestive as to the method of treating the twisted loop of bowel in certain cases. Mr. Turner quite agreed with the remarks of Mr. Cripps as to the length of incision. He thought a small incision almost useless. In so many cases the diagnosis of the form of obstruction was very obscure and difficult. He had seen a case in the *post-mortem* room where the exact details of the obstruction were made out with difficulty. He would not hesitate, in cases of distension of the gut, to evacuate the contents in the manner practised by Mr. Cripps. In conclusion, he thanked the Society for the way his paper had been received.



## THE TREATMENT OF THE PERITONEUM IN ABDOMINAL SURGERY.

By W. A. MEREDITH, M.C. Edin.

THE attainment of success in abdominal work, as in any other department of surgery, must naturally in great measure depend upon the training and special experience of the individual operator. But apart from these general factors the observance of certain important details in relation to the methods of treatment employed undoubtedly tends to influence the proportion of successful results obtained in any given instance. Surgical procedures implicating the peritoneum may in some degree be considered as regulated by different laws from those which guide the surgeon in dealing with other serous cavities. This fact is dependent not alone upon the large extent of the peritoneal surfaces, with their numerous folds and recesses, but, further, upon the peculiar vital properties which characterise this membrane, as regards both its special structure and its intimate relation to the main excretory organs of the system, through the ready channel afforded by the subjacent lymph and blood vessels. In evidence of the enormous powers of secretion and excretion possessed by the peritoneum, so long as its epithelial surface is comparatively intact, one has but to consider the readiness with which effusion of so-called ascitic fluid takes place as a result of interference with the return of blood through the portal system; and, again, the equally wonderful rapidity with which such effusion may disappear upon the re-establishment of the normal balance of circulation within the vessels underlying the serous membrane; whether as the result of removal of the original hepatic obstruction, or as a consequence of relief to the vascular engorgement obtained by derivative action through the renal and intestinal emunctories.

With these facts in view, and therefrom considering the peritoneum in the light of a huge lymph sac, one cannot, I think, too highly estimate the practically unlimited powers for good as for evil which it possesses in relation to operative measures involving its cavity. From such a standpoint as this, it seems to me that success in abdominal work, apart from the general factors already



alluded to, may be said to largely depend upon the attainment by the surgeon of three main objects, which should be kept constantly in view when dealing with the peritoneal cavity.

*First* of these three I will place "avoidance of the introduction into the serous cavity of any septic or potentially septic material," comprising under this latter denomination any substance or fluid which may, under favouring circumstances, afford a protective nidus or medium for the subsequent development of organisms within the peritoneal sac. It may, I believe, be definitely asserted that the surest and most effective protection against the risk of such introduction of mischief from without during abdominal operations lies in the rigid routine employment of antiseptic—preferably carbolised—lotions for sponges, ligatures, and instruments, as well as for the hands of the operator, his assistant, and nurses. While expressing this opinion, the truth of which is, to my mind, absolutely certain, I am fully prepared to admit the possibility of the attainment, in skilful hands, of good results without the use of any antiseptics whatever during intra-peritoneal operations, providing always that scrupulous cleanliness of procedure be observed, and that the recognised methods of flushing and subsequent drainage of the abdominal cavity be followed in suitable instances. But, at the same time, I have no hesitation whatever in stating that a surgeon who thus trusts to the use of plain water as a substitute for carbolised lotions unwisely neglects one of the surest safeguards against septicæmia, and that, although he may for a while secure good results, his work, in the long run, must inevitably suffer from such omission. The explanation of this fact is not far to seek. It is not possible, even with the strictest use of antiseptic methods, to entirely banish all risk of septicæmia from abdominal work, since one is constantly exposed to meeting with cases where the poison in an active or potentially active state is present within the peritoneal cavity before operation—*e.g.*, ovarian cysts with septic contents as a result of previous tapping, putrid gestation sacs, tubes distended with puriform fluid, pelvic abscesses, &c. Under such conditions, a free use of antiseptics, with the object of minimising the risks of subsequent mischief by weakening the already existent germs of infection, and thus rendering them an easier prey to the destructive power of the leucocytes, offers indisputable advantages over those afforded by the employment of plain water for mere cleansing purposes.

A further advantage possessed by a Listerian operator—one more readily appreciable, perhaps, in hospital than in private practice—is the degree of protection afforded by the routine use of antiseptics against the recurrence of disaster after a death from septicæmia. Anyone who will take the trouble to examine the record contained in Sir Spencer Wells's work on 'Ovarian and Uterine Tumours' (London, 1882) of consecutive ovariectomies performed previously to his adoption of the antiseptic method in 1879, cannot fail to observe that a death from septicæmia, occasionally succeeding to a series of as many as twenty-five recoveries, was almost invariably followed by a more or less interrupted succession of fatalities attributed to septic causes. Such a noteworthy fact—accountable for only by the transmission of infection from one case to another, whether through sponges, silk, instruments, or by the hands of the operator or nurses—constitutes, to my mind, one of the strongest arguments in favour of antiseptics in abdominal work, since we know that by their use we can surely lessen, if we cannot altogether avoid, the risk of thus unwittingly conveying infection from one patient to another.

Turning now to the *second* of the objects to be kept in view by the abdominal surgeon, I would define it as "avoidance of the infliction of any unnecessary injury to the peritoneum, whereby its vital properties may be impaired." It was long ago pointed out by Sir Joseph Lister that any suspension of vital activity in a tissue as the result of injury leads to paralysis of its special functions and to consequent impairment of its ability to resist the entrance of parasitic organisms. This observation is one of very grave moment when considered in relation to its bearing upon so delicate a structure as that which characterises the serous membrane lining the abdominal cavity, and in confirmation of its importance we have the statement of so high an authority on this subject as Professor Watson Cheyne, to the effect that "one of the conditions favouring the development of peritonitis as a consequence of the introduction of cocci into the peritoneal cavity is an abnormal state of the serous membrane whereby its absorptive power has been diminished—as, for example, by the action of any substance which weakens or kills the serous tissue, and thus provides a suitable soil for the penetration of the cocci."\*

Now depression of vitality may be either general or local; and

\* 'Brit. Med. Jour.,' vol. i, 1888, p. 524.



any method of treatment tending to induce it in either direction should, in my opinion, be sedulously avoided. This brings me to the question of the employment of the carbolised spray in abdominal work. I have myself entirely given up the spray since the end of 1888; and, so far as I know, its use has now been abandoned by the leading abdominal surgeons in this country, with the sole exception of Mr. Thornton, who, I believe, still employs it in peritoneal operations. Were it not for this latter fact I should not consider it necessary to further allude to this subject of the spray, but under the circumstances I deem it well to briefly note my own chief objections to its use. In the first place, we have no proof whatever that the carbolised spray can safeguard us against the introduction of septic mischief into the open abdominal cavity otherwise than by maintaining a form of constant antiseptic irrigation for moistening the exposed surface of the patient's abdominal wall, as well as the hands of the operator, his assistant, and nurses. While fully admitting its usefulness in this respect, I am convinced that the object in view can equally well be attained by the exercise of ordinary care on the part of a Listerian operator in the way of sponging and frequent ablutions; and that he may thus secure all the advantages derivable from the use of carbolic acid without exposing his patient to the risks entailed by the chilling properties of the spray. These latter constitute my own chief objection to its employment. Anyone who has stood for an hour or more assisting with bared arms in the performance of an abdominal section under a steam spray can form for himself a tolerably accurate estimate of its powers in this direction; and thus in some measure conceive the extent of its depressing influence upon the vitality of the patient, a portion of whose abdominal wall is of necessity exposed to the cold moist blast. Such exposure during prolonged operations, especially in wintry weather, undoubtedly entails serious risk of death from shock in the case of feeble, elderly patients; while, short of this, it not unfrequently interferes gravely with the progress of convalescence by originating troublesome chest complications. In addition, however, to the depressing influence of the steam spray upon the general vitality of the patient, one must further take into account its direct action upon the peritoneum itself during the removal of large solid tumours necessitating a lengthy abdominal incision, or in cases of ovarian or other cysts



complicated by the presence of extensive adhesions. Under such circumstances I am convinced that prolonged exposure to the chilling and irritating influence of the carbolised spray—while necessarily increasing the general depression—undoubtedly further tends to temporarily impair the vitality of the serous membrane itself over areas of greater or less extent; and, by thus lessening its powers of absorption, to induce the abnormal condition above referred to as favouring the penetration of cocci and the consequent development of peritonitis.

A still more definite, but commonly unrecognised, source of injury to the peritoneum, leading to more or less extensive impairment of its vital functions, arises from the performance of what was formerly known as the “*toilette du péritoine*”—a procedure commonly involving assiduous and prolonged sponging of the abdominal cavity in cases of extensively adherent or ruptured cysts.

In my early operating days I naturally followed in this regard the example set me by my two senior colleagues at the Samaritan Hospital, both of whom were then extremely careful in thus cleansing the peritoneal cavity at the conclusion of an abdominal operation. Such treatment, in complicated cases, was usually followed within forty-eight hours by a smart rise of temperature to  $102^{\circ}$  or over, frequently necessitating the use of an ice cap for its reduction. When I began in 1882 to assist Sir Spencer Wells, who then operated with full antiseptic precautions in every respect similar to those employed in the Samaritan Hospital, I soon became impressed by the fact that complicated cases of ovariectomy, where no attempt was made to sponge away the mixture of blood and other fluids remaining in the pelvic cavity at the conclusion of a troublesome operation, not unfrequently recovered with but very slight disturbance of either temperature or pulse, although no drainage-tube was used. Such results, following treatment so opposed to the routine practice in the hospital at that time, puzzled me somewhat, until I formed the conclusion that avoidance of unnecessary irritation of the serous membrane might have something to do with them; and, as time went on, my views in this direction were materially strengthened by watching the results obtained in cases where the abdomen was washed out with warm water instead of being cleansed by sponging. I have now myself employed this treatment of “flushing” with increasing

frequency for over four years past, and am constantly more and more impressed by its value—as a ready and efficient means of cleansing the peritoneal cavity with the minimum infliction of damage to the delicate epithelial surface of the serous membrane—a fact which, from my point of view, constitutes one of its greatest advantages. As a firm believer in the importance of asepticism, I need hardly add that I consider it advisable always to employ for this purpose water which has been previously sterilised by boiling, as the surest means of avoiding the introduction of possible mischief into the peritoneal cavity.

Passing on now to a brief consideration of the *third* of the three objects to be aimed at by the abdominal surgeon, I define it as the “promotion of the subsequent removal by natural or by artificial means of any fluids remaining in the pelvic cavity at the conclusion of the operation.” By the term “natural means” I would imply the early re-establishment and maintenance of the renal and intestinal eliminatory functions which play so large a part in promoting the absorption and subsequent excretion of peritoneal effusions. Without dwelling on this subject I will here merely refer to what I believe to be the extreme inadvisability of the routine administration of opium after abdominal operations, owing to the restraining influence thereby exercised upon these functions; while further noting the value of repeated small doses of salines administered, not with a view to relieving any tendency to obstructive bowel difficulty, but merely as promoting renal and intestinal excretion. By the artificial or mechanical means above mentioned I, of course, allude to drainage of the pelvic cavity as best effected by the ordinary glass tube, whence the accumulated fluid is withdrawn at stated intervals. An objection formerly raised against this method of drainage was based on the statement that it was liable to be followed by a ventral hernia at the seat of insertion of the tube. Such a mishap has fortunately been extremely rare in my own experience, and I regard its occurrence as indicative of too prolonged retention of the tube. In my opinion, all the good derivable from drainage of the peritoneal cavity proper is commonly attained within the first twenty-four to forty-eight hours after operation, and any further prolongation of the process is but rarely advisable. My own invariable rule is to remove the tube whenever the entire amount of fluid accumulated during the previous twelve hours is found not to exceed 2 to

4 drachms, and this quite irrespective of whether the serum then withdrawn be blood-stained or not.

In support of the views here expressed, I will now very briefly summarise the results obtained in my practice from December, 1888, when I abandoned the use of the spray, to the end of July, 1892. During this period of about three years and a half, the total of my abdominal operations amounted to 201; but in 18 of these, chiefly cases of pelvic abscess, the serous cavity proper was not invaded,\* and I shall consequently confine my remarks to the remaining 183 operations, all of which implicated the peritoneum. Of this number, 42 were undertaken for disease either unconnected with the pelvic organs, or, when so connected, of such a nature as not to admit of complete extirpation. 33 of these patients recovered, and the remaining 9 died from causes shown in the accompanying table.

TABLE I.—*Peritoneal Operations.*

No.	Nature of disease.	Cause of death.
1.	Double intra-peritoneal abscess (of septic origin).	Shock, within six hours.
2.	Cancer of both ovaries (partial operation).	Shock, within four hours.
3.	Advanced tubercular disease of peritoneum.	Exhaustion on twelfth day, from persistent diarrhoea.
4.	General abdominal cancer.	Exhaustion, on eighth day.
5.	Pelvic cancer.	Exhaustion, on fourth day.
6.	Intra-peritoneal abscess (? of tubal origin).	Exhaustion, on seventh day.
7.	Retro-peritoneal sarcoma.	Exhaustion, on third day.
8.	Cholecystotomy.	Perforation of intestine, on twenty-eighth day.
9.	Hydatid disease of liver.	Cerebral disease, on eighth day (? hydatid).

The remaining 141 peritoneal operations were performed for disease originating in the uterus or its appendages in the following proportions, and with the results here shown—viz., 137 recoveries and 4 deaths.

\* The group of eighteen operations above referred to included seventeen recoveries and but one death, which resulted from exhaustion due to diffused pelvic suppuration of some two years standing previous to surgical interference.



TABLE II.—*Peritoneal Operations.*

Nature of disease.	Cause of death.
Eighty-four cases of ovarian tumour.	One death from pneumonia on thirteenth day.
Fifteen cases of chronic inflammatory disease of one or both uterine appendages.	One death from intestinal obstruction on eighth day.
Five cases of ruptured tubal pregnancy with intra-peritoneal hæmorrhage.	One death from exhaustion within five hours.
Thirty cases of supra-vaginal hysterectomy for fibro-myoma.	One death from shock.
Seven cases of removal of the uterine appendages for fibro-myoma.	No death.

This series of 141 operations, performed on the lines indicated in my paper, with a mortality of under 3 per cent., may, I think, be taken as affording some evidence of the correctness of the views advocated with regard to the treatment of the peritoneum in abdominal work; and, in conclusion, I would here venture to express the hope that the record of my experience in this direction may tend to promote a fuller recognition of the enormous powers for good as for evil possessed by the serous membrane in relation to surgical procedures involving its cavity.

Mr. ALBAN DORAN noted how Mr. Meredith spoke of "unnecessary injury of the peritoneum." Although so much had been written about peritonitis, the limits of that disease had not been clearly defined. Long experience had led Mr. Doran to believe that it was not always possible to distinguish between the effects of damage to the peritoneum and the effects of damage to the intestine caused by exposure, handling, and bruises from instruments. Great importance must be attached to tympanitic distension after abdominal section. Mr. Tait held that it was the result or chief sign of peritonitis, Mr. Malcolm considered that it was a primary condition. There could be little doubt that tympanites was one of the cardinal symptoms of peritonitis; nor could there be much doubt that it was in many cases a symptom of damage to intestine acting through nerves and vessels. The intestine was paralysed, and the patient sometimes died, no evidence of peritonitis being found after death. Mr. Doran asked for an explanation of the beneficial results which seemed to follow simple section of the peritoneum in exploratory operations. Disseminated papillomata sometimes disappeared. He referred to a case in his own practice ("Anterior Serous Perimetritis," 'Trans. Obstet. Soc.,' vols. xxxi, xxxiii), where he made an incision through the abdominal walls into a spongy vascular mass, which simulated a large malignant cystic tumour. The entire "tumour" disappeared, and the patient lived over three years, dying of phthisis. The appendages showed signs of old tubercular disease. Mr. Doran objected to Mr. Meredith's practice of

removing the drainage tube whilst bloody fluid still issued from it. Klotz's researches ('Centralblatt für Gynäkologie,' No. 29, 1892) showed that one distinct cause of intestinal obstruction (and this brought us back to the question of tympanites) was the adhesion of intestine to a large organised clot. The more the blood came away the less danger would there be of the development of a clot large enough to do harm. Not long ago Mr. Doran removed the drainage tube whilst bloody fluid was still escaping in small quantities. Troublesome symptoms of obstruction followed almost immediately, and were not overcome without difficulty. He entirely agreed with Mr. Meredith as to flushing, a practice for which, it is fair to remember, we were indebted to Mr. Lawson Tait. It acted partly as a hæmostatic and partly as counteracting shock by warming the abdominal cavity. He had noted shock when the injected water was too cold. French observers had shown that flushing also fed the patient. Several pints at least were usually absorbed, as the operator never allowed all to return through the abdominal wound. The French encouraged absorption by adding table salt to the water used for flushing.

Mr. MALCOLM said he had come to a somewhat different conclusion from that of Mr. Meredith as regards the delicacy of the peritoneum. He believed that so long as no septic contamination was allowed the peritoneum would tolerate without difficulty any manipulation that was necessary. He pointed out that Mr. Meredith had himself recorded in his paper that cases in which no effort was made to clean the peritoneum, and no drainage was used, had frequently astonished him by their perfectly smooth recovery. On the other hand, Mr. Malcolm had seen, and Mr. Meredith must also have seen, the peritoneum treated in the roughest manner, literally scrubbed with sponges, the intestines being freely brought out of the abdominal cavity, and yet the convalescence had been absolutely as smooth as one could wish. It seemed almost as if one could do as one liked with the peritoneum, and leave a certain amount of clot and serum inside with impunity. But there was one point in these cases of abdominal surgery, to which Mr. Doran had kindly alluded, and on which Mr. Malcolm laid the very greatest stress, namely, the importance of avoiding distension of the intestines during convalescence. Mr. Lawson Tait had astonished the profession, about the year 1887, by announcing that he cured peritonitis after abdominal section by purgatives. Mr. Malcolm did not believe that Mr. Tait had ever done anything of the kind. He had merely cured slight obstructive conditions, and so prevented the occurrence of tympanites and consequent peritonitis. Mr. Malcolm maintained that all the recent improvements in the treatment of cases of abdominal surgery are directed to the avoidance of this intestinal obstruction; that starvation by the mouth acts in this way by resting the bowel and giving it time to recover from the manipulation to which it has been subjected; that the old treatment by a constant use of opium bound up the bowels, and aggravated any tendency to obstruction which might exist, while a more judicious management in this respect may obviate the danger; that in the same way purgatives and purgative enemata act favourably by keeping the alimentary canal open. As regards washing out the peritoneal cavity, the beneficial influence was attributed by the speaker in great part to the perfect way in which this process accomplishes a favourable disposition of the disturbed intestines by first floating them up, and then allowing them to settle down quietly and naturally. There could be no doubt that one of the most



important points in the management of these cases was to get the flatus to pass down and thus to avoid tympanites. When flatus passed down freely the patient very usually got well, and this question could not be left out in considering the treatment of the peritoneum in abdominal surgery.

Mr. MARMADUKE SHEILD remarked on the difficulty in some cases of preventing the peritoneum from being stripped up from off the anterior abdominal wall, and to prevent this he usually stitched it provisionally to the skin before proceeding to manipulate the abdominal cavity.

The CHAIRMAN (Dr. Symes Thompson) remarked that in the rooms of the Medical Society of London, since he became a Fellow in 1859, the evidence adduced in favour of abdominal section had advanced in a truly remarkable manner. In earlier days operations were too often delayed until it was too late, whereas at present, and under antiseptic precautions, the operation was found to be so free from risk that there was a danger lest it should be resorted to unnecessarily. The change that had been wrought in thoracic as well as in abdominal surgery deserved the fullest recognition, and in calling on Mr. Meredith to respond the Chairman expressed the thanks of the Society for his valuable contribution.

Mr. MEREDITH, in replying, stated that his practice in relation to antiseptics had not varied since he first began to operate thirteen years ago, excepting as regarded the use of the spray and the adoption of the "flushing" treatment. He had always remained a consistent advocate of the Listerian system. Referring to Mr. Marmaduke Sheild's remarks, he was of opinion that the stripping of the peritoneum off the anterior abdominal wall in cases of very firmly adherent cysts was not usually an accident of serious moment, providing that the operator realised his mistake in time to avoid any extensive denudation of the under surface of the *rectus* sheath, and was subsequently careful not to leave any blood-clot between the peritoneum and the muscle layer when closing the abdominal incision. The chief motive of this paper was to show the importance of leaving the peritoneum at the conclusion of an abdominal operation in as normal a condition as possible, with a view to facilitating the subsequent performance of its natural functions. The exceptional illustration of the good effects obtained by assiduous sponging, coupled with removal of the effused serum in certain cases of tubercular disease of the serous membrane, was explained by the resulting impaired nutrition of the morbid growth, and this fact afforded confirmatory evidence of the extreme susceptibility of the peritoneum to the influence of rough treatment.

---



*October 31st, 1892.*

## INTRA-THORACIC AUSCULTATION AS A MEANS OF PHYSICAL DIAGNOSIS.

By BENJAMIN WARD RICHARDSON, M.D., F.R.C.P., F.R.S.

MR. PRESIDENT AND GENTLEMEN,—I had promised to bring before the members of the Medical Society of London this session a paper on Some New Researches in Synthetic Pathology, but as I was not prepared for so early a call for the paper as that which has been assigned to me I have been unable to complete it in the form in which I would wish to present it. It happens, however, that I have at hand a short communication which I have much pleasure in submitting to you, and which, because it is extremely simple and practical, will, I trust, be accepted as a substitute for the longer and more ambitious essay. I call the present essay a study of Intra-thoracic Auscultation as a Means of Physical Diagnosis, and I cannot introduce it to your notice better than by relating how it came into my mind and practice. A few months ago a patient consulted me who was suffering from serious and obscure symptoms referable, by the process of exclusion in diagnosis, to the upper portion of the alimentary canal. He had lost flesh to an extreme degree, was very feeble, had often a difficulty in swallowing food, at times retained food of a fluid or semi-fluid kind in the stomach for long periods, and then after suffering severe pain vomited it with difficulty, returning it in a partially digested state. I looked upon the symptoms with suspicion as possibly indicating malignant disease of the lower part of the œsophagus; but as I found he had been following an imprudent dietary I was content at the moment to regulate diet carefully and to prescribe a mixture of dilute hydrochloric acid and pepsine. He left me, to return in two months reporting himself in every respect better. He had gained in flesh, he retained food, was free from acute pain, had improved in strength, and had lost the sense of weariness of mind as well as body, which had been most oppressive. He had determined to take a holiday, and I agreed with him that the change he suggested would be advantageous. I did not see this patient again for three months, when he con-

sulted me once more in consequence of a sudden return of his worst symptoms, to which were added others pointing more decisively to œsophageal mischief low down in the tube. With difficulty he had partaken of a rather too copious meal one day previously, and soon afterwards had been seized with acute pain, which lasted until the undigested mass had been vomited with free secretion of the gum-like mucus characteristic of stricture. He was again greatly emaciated, presented a condition of circulation so feeble that I could scarcely detect the radial pulse, and a heart so weakened it was difficult to distinguish clearly the two sounds.

I tried in this case what I have called the water-gurgle test for the diagnosis of stricture, as described in 'The Asclepiad,' vol. vii, p. 332—that is to say, I got the patient to attempt to swallow fluid whilst I auscultated in the line of the œsophagus anteriorly and posteriorly. Whenever there is true stricture of the organic type I have usually found by this method a point where there is heard a loud gurgling sound on attempts to swallow, followed by a sharp noise as of a passing current of fluid through a constricted passage. There are few more characteristic points of diagnosis of stricture in the lower third of the tube than this; but there was no response to the test in the present instance, and the patient expressed to me that the tumbler of milk-and-water which he had swallowed went down without his being conscious this time of obstruction. I turned, therefore, naturally to the use of the œsophageal tube—an operation which led me to the new facts I have to record. I passed along the œsophagus a medium-sized tube, and ran it without difficulty down to the stomach. There was no serious obstruction at any part, but I thought I experienced some sense of friction of a very slight kind. Whilst endeavouring to be certain on this matter an idea which I had once before had in my mind, but had not before acted upon, suddenly occurred to me. Why not auscultate through the exploring tube? At once I sliced off a portion of the free end of the tube obliquely, slipped over this sliced end the terminal part of a double stethoscope, and made in this fashion the exploring tube a continuous stethoscope. The effect of auscultating in this way was most interesting and satisfactory. I could hear soft friction of the tube against the walls of the œsophagus, and was made quite sure that the friction was uniform throughout and that there was no special



constriction or induration in any portion of the tube. When I passed the tube into the cavity of the stomach itself I obtained a sound new to me, like a gentle seething as of air or gas agitated in a thickish fluid; and, at times, a gurgling sound of gas with another sound probably due to muscular contraction of the stomach itself. As the patient experienced no trouble or inconvenience during examination I had ample time for inquiry, and I leisurely withdrew the tube, noting the sounds audible in the course of the movement. In the tube at this time there were only two openings and those at the extreme end. I succeeded therefore in catching sounds at such points only as were in apposition to the openings. I withdrew the tube until the opening on the left side came in contact with that portion of the œsophagus that lies in immediate proximity with the heart. By previous auscultation of the heart over the thoracic wall I had failed to detect clearly the two cardiac sounds owing to the feebleness of the cardiac action, but now both sounds were as distinct as they would have been from a normal heart. They were not, however, precisely the same as the sounds we hear through the thoracic wall; they were duller in character, as if they wanted the resonance which is probably produced by the pleura stretched over the thoracic cavity. At the same time they were loud and were singularly distinct. By moving the tube gently up and down I could get the second sound separately from the first and *vice versâ*; but when I had the opening of the tube midway so as to compass both sounds, there was not so much difference between the first and second sound as is common to that distinguishable in ordinary auscultation. I was quite prepared for all these modifications of phenomena; they corresponded precisely with what I had learned many years ago when, in combination with the late Drs. Baly and Sibson, I had seen Dr. Halford demonstrate Brier's valvular theory of the cause of the two sounds. We listened at that time directly to the sounds from an opening in the chest wall of a lower animal under anæsthesia, and detected that with such immediate auscultation the sounds were deficient in sharp resonance, and more equable in tone than was common from ordinary auscultation. It was the same now. I counted the beats of the heart very deliberately from the inside of the thorax, seventy beats per minute, the sounds and the pause in proper order, and the action perfectly regular. I expected that on withdrawing the tube further out of the œsophagus



it would be possible to hear a loud sibilant or vesicular murmur in respiration. In this I was disappointed to a certain extent. It was impossible, even on a deep inspiration, to catch a murmur so distinct as the murmur heard from the chest wall outside.

From these observations I have been led to the new departure in physical diagnosis in which I am anxious others should take part, and I have devoted some time to certain preliminary steps in its development. Briefly it is a means for auscultating on an extensive scale the organs of the body *from within the body*. I shall occupy most usefully the short remaining time at my command, first, by indicating the lines of research in which the plan promises to be most useful; second, the limitations of the plan and, if I may so express myself, the objections to it; and, third, the modes by which it may be improved from this its original start, so as to make it ready, safe, and in its broadest sense useful.

Touching the first of these points, the method promises to be useful—I hope very useful—in the diagnosis for which I first applied it. It proved of service to me; at the moment it told me distinctly, in the case I have referred to, that there was no constriction, no induration, at any part of the œsophagus, and that the hearing confirmed the touch, or, rather, corrected it in a manner that could not have been more satisfactory. The advantage will be that stricture may be detected in its very earliest stage—the stage in which, according to my experience (unfortunately an experience specially large in this disease), there is the only chance of doing good by dilatation. The new auscultation may prove also an aid to diagnosis in diseased conditions of the stomach itself. It is certain from the sound I heard from within the cavity of the stomach that there are going on there changes of a physico-chemical kind, leading to a sort of effervescence that is distinctive in character. It is most probable there are differences of sounds connected with special fermentations, and that by study and experience these differences may become detectable at once by auscultation. Here there is an open field for research in which I have been unable as yet to enter. Again, as bearing on the stomach, the motions of that organ evidently produce sound that will admit of interpretation. A healthy stomach may possibly give forth no sound or it may give forth a definite sound when containing food, with other sounds during various stages of digestion.

These are points to be inquired into and will be of curious interest. In diseased conditions of the stomach a whole series of diagnostic symptoms may also be learned, bearing not only on fermentative actions in progress, but on alterations in the walls, and on its contractile functions. The stomach, in short, will admit of being sounded like the chest. Scirrhus affecting the stomach should be easily diagnosed by auscultation. Through the stomach it may also be possible to diagnose physically and more correctly than we now diagnose the nature of some pulsating abdominal tumours which are, as they have been since the time of William Harvey himself, a constant source of disagreement among physicians. With the terminal of a full-sized œsophageal stethoscope in the stomachic cavity a loud murmur from an arterial source will be detected without the interposition of pressure and an important difficulty in diagnosis removed. A third service that may be rendered by intra-thoracic auscultation is forecast in the observation I have already made respecting the detection of the cardiac sounds in conditions when those sounds are inaudible under the usual method owing to extreme feebleness of the circulation. We are called sometimes to persons in such entire collapse that it is doubtful whether they are alive or dead. The sounds of the heart are imperceptible to the ear through the thoracic walls. Here, then, is an instant and ready method of deciding whether the action of the heart is still in progress. We have a new proof either of absolute death or of continued life. In acute collapse, as after death from chloroform, we not only can institute a good diagnosis in the same manner, but we are half way, by our manipulation, towards assisting to restore life. Through the œsophageal tube, after disconnecting the stethoscopic tubes that go to the ear, we could inject a free quantity of heated water or water with oxygen peroxide into the stomach; or, if we had learned the practice correctly, we could direct a faradaic current upon the heart itself, so as to excite contraction of the right side.

The use of the intra-thoracic method may be turned to account in diagnosis of heart disease and of aneurysm of the large thoracic arterial trunks. Stricture of the œsophagus arising from aneurysmal pressure would be instantly diagnosed. These cases are often most perplexing. The pressure causing the obstruction varies, and no sufficient sign of aneurysmal murmur in the early stages reaches the ear at all times. One auscultator fortunately



catches the murmur, another does not, and so contradiction upon contradiction amongst the best diagnosticians occur. By the process of intra-thoracic auscultation this difficulty would be immediately met, the pulsation would come under direct observation, and the precise seat of the pressure would be descended upon. We should hear a pulsating stricture. In heart disease itself the intra-thoracic mode of examination should be of service. By it in cases of enlargement we ought to be able to distinguish between dilatation and hypertrophy. The diagnosis of clot on the right side might be made clear by this plan and the differential diagnosis of valvular affection on the right or left cavities ought to be rendered absolute, when we have learned sufficient of œsophageal auscultation to discover the new distinctions of sound that will have to be made, in some cases at least, between the tone of the first and the second sounds, and shall have defined all new lines of distinction between these sounds as heard from within and from without the exterior walls of the chest. The clearest definition by this test should be obtainable also as between pericardial and endocardial friction sound and between pericardial as distinct from pleural friction.

As this is only a preliminary note, I must leave untouched the subject of pulmonary auscultation by the intra-thoracic method, and also that of pharyngeal exploration. The pharynx is within such easy reach it should admit of more frequent auscultation than lower parts of the œsophageal canal. Its size, too, renders it more accessible. From it we should be able to reach the apices of the lung and the greater part of the air passages from the larynx down to the bifurcation of the bronchi. Exploration of these parts will be easy when a set of appropriate and convenient exploring instruments have been constructed, and will afford help to diagnosis of changes incident to the apices of the lungs in sub-clavian, innominate, and carotid murmurs, and in thickenings and obstructions of the trachea and larynx.

Having given an outline of what may be gained by intra-thoracic auscultation, I shall now refer to the limitations of the plan and to the objections which may be taken to it. I assume at once that this mode of research is not called for when by the ordinary auscultation diagnosis is clear. It may come in usefully in all cases where the œsophageal tube is subjected to exploration, but it will not be always applicable in instances where it might be useful,



owing to the circumstance that many patients are unable to bear the introduction of the tube and that it may sometimes be unadvisable to subject them to it. I met with a patient last week suffering from probable thoracic aneurysm in whose case the exploration would have been most valuable; but the introduction of the tube caused so much retching and straining I was obliged to withdraw the tube before I had explored as fully as I could have wished, although what was effected was sufficient to inform me of the existence of aneurysmal murmur from the left carotid or subclavian. The explorations might also be objectionable in cases of irritable stomach or where there was severe cough or cough with hæmorrhage. I need not dwell any longer on particular points of this kind, since the whole is summed up in a sentence—whenever the practitioner feels he can safely and prudently pass a tube into the stomach, then he can bring into practice intra-thoracic auscultation.

The third point, the modes in which this method may be improved by mechanical means, opens up a very wide field of inquiry. I commenced my work with the ordinary flexible tube, and up to the present time I have found nothing better than a good-sized tube with a large lateral aperture at the extreme end. I have used another tube charged with several apertures an inch from each other, and this answers fairly well. Apertures are essential in these tubes; if they are not made there is little or no conduction of sound. Messrs. Krohne and Sesemann have been making tubes for me of different materials; and one of metal, of malleable nature, is good in many ways, but I have not yet obtained what is precisely wanted even in so simple an invention as, at first sight, it would appear to be. The best tube at this moment is the long œsophageal flexible tube with the stethoscope attached as shown to-night.

I leave now this contribution in the hands of a Society to which it has been my high privilege to communicate many of my first thoughts from the very opening of my long career. I would not on any account attach an undue importance to the effort, or look upon it as anything more than an extension of the simple act of the illustrious Laennec, when, in the Necker Hospital one day in 1816, he improvised a stethoscope out of a roll of paper and projected thereon a new science to which there seems to be no end. I put forward this addition, however, without apology, not knowing

to what—small as it is at this precise moment—it may lead. In his magnificent eulogy on his master, Cullen, Benjamin Rush said : “Let no fact in medicine, however unimportant it may at first appear, be allowed to pass unnoticed by the public eye ; for there are mites in science as well as in charity, and the ultimate results of each are often alike important and beneficial.” In that spirit, Sir, I offer to-night, through you and my fellow colleagues of the Society, one more mite to the treasury of practical medicine.

Dr. ROUTH, after complimenting Dr. Richardson on his very interesting and original paper, said he was not sure that it would be received with good will by all members of the profession. Conservative as he was in politics, he feared there would always be persons in the profession too conservative in their views to welcome Dr. Richardson's new departure. *Intra-audition* was not popular in the profession. He, himself, had years ago read a paper before the British Medical Association, which was very well received, in which *intra-audition* on the caudal side had been proved to be very efficacious in diagnosing gestation. He had used a binaural stethoscope with a long glass tube at the distal end, which after a series of experiments he found the best form for use. When this was inserted into the vagina, you could hear directly the uterine sounds of pregnancy. In very early periods it was a continuous murmur not unlike that heard in an enlarged spleen. In a month this sound was interrupted at long intervals ; in six weeks you could hear the placental souffle, and later on the foetal heart when you could not do so by auscultation over the abdomen. Nothing could be more delicate than its use, as all could be done under the clothes, the glass occasioning no friction. Yet but few used it, although it had been of the greatest use in doubtful cases. Dr. Richardson must be prepared for opposition, therefore, and yet he felt that he had made a very remarkable innovation in practice, and one likely to prove of the greatest advantage.

## THE SURGICAL TREATMENT OF CYSTS OF THE VULVO-VAGINAL OR COWPER'S GLANDS.

By ALBAN DORAN, F.R.C.S.

A. H., aged 48, single, came under my care early in January, 1890, complaining of a swelling in the external parts, which had been noticed for several months, and caused great pain whenever she walked or sat down.

An oval fluctuating tumour, about  $2\frac{3}{4}$  inches in long or antero-posterior diameter, occupied the position of the left labium majus, pushing forwards the labium minus. The tumour reached the



mons veneris, but was not connected with the inguinal canal; it was irreducible and there was no impulse when the patient coughed. Posteriorly it ended, as a blunt-pointed process, in the left ischio-rectal fossa. It did not extend into the vagina. The integument was inflamed on both sides of the tumour.

The patient was put under chloroform. I made an incision, about 1 inch long, through the skin, antero-posteriorly, nearer the vulvar than the outer limits of the cyst. The surface of the cyst was white; it gave way and over 6 ounces of a thick, greasy, orange-brown semi-fluid material escaped. I dissected the upper and anterior parts of the cyst away from their connections with little difficulty, except that at one point, where the cyst-wall was inflamed and degenerate, it adhered firmly to surrounding structures. I passed my left forefinger, behind the pointed posterior extremity of the cyst and dissected it away. This part of the operation was not easy, as the patient took chloroform badly. When she began to breathe well, I separated the remaining connections which lay in the direction of the transversalis perinei artery. Four branches were divided and bled freely, but were easily secured and ligatured. As usual, the posterior part of the cyst lay much deeper than might have been expected from superficial examination.

In order to check oozing, which was free, I syringed out the wound thoroughly with hot water, packed into the pouch a roll of absorbent gauze, and united the integument along the anterior two-thirds of the wound with silkworm-gut sutures. A pad of absorbent wool was placed against the labium and fixed there by means of a T-bandage.

Five hours later I found that much blood had soaked through the dressings; no blood was then oozing from the wound. I placed a clean piece of absorbent gauze in the wound, and on the next day removed it, syringed out the wound, and inserted a piece of red rubber tubing. This was removed in a few days, and the wound healed perfectly. Six months later there was no trace of any fistula.

The cyst, which is now exhibited, is thin-walled, and, after having shrunk in spirits, measures over  $3\frac{1}{2}$  inches in its long diameter. It is unevenly divided into two portions by a deep constriction. The anterior is much the larger, and measures over 2 inches at its widest part. The posterior is the blunt-pointed process which was dissected out of the ischio-rectal fossa. Its inner wall bears



incomplete septa, whilst in the anterior part of the cyst that wall is almost smooth. The outer wall of the cyst bears portions of muscle, fat, and other tissues with which it was closely connected. Some microscopic sections were prepared at the College of Surgeons. The cyst-wall was thick, and chiefly made up of very pure white fibrous tissue. It contained a considerable number of arterioles with thick muscular walls. The inner aspect was devoid of epithelium. This specimen now belongs to the Museum of the Royal College of Surgeons. (Pathol. Series, No. 4688B.)

From its position, it seems clear that this cyst was developed from the vulvo-vaginal gland, which is often known by the names of Cowper, Bartholin, and Duverney. In some respects, "Cowper's gland" is the best name, as it is the same as that of its homologue in the male, which cannot be called "vulvo-vaginal." The anatomy of the gland is well known. The duct pierces two layers of pelvic fascia. This reminds us that the gland is deeply placed. The orifice of the duct lies external to the hymen, and is not hard to detect in many subjects.

The pathology of these cysts is of distinct surgical importance. Other kinds of vulvar cyst must be noted. First, there is the form corresponding to well-known conditions in relation to inguinal hernia in males. In 1884 Dr. Galabin and Mr. W. E. Fielden exhibited at a meeting of the Obstetrical Society of London a "Cyst removed from the Vulva." It lay in the left labium majus of a girl aged 14. Anteriorly, it ended at the level of the clitoris, where a firm cord seemed to connect it with the inguinal ring. Hence it was believed to be a hydrocele originating in a pouch of peritoneum. The cyst now exhibited was not of this class, as the disposition of the pelvic and perineal fasciæ would prevent a process of peritoneum from burrowing into the ischio-rectal fossa.

There is at least one class of cyst decidedly not of peritoneal origin, nor developed from the vulvo-vaginal gland. There are great discrepancies in the pathology of this class amongst authorities; possibly two or more varieties have been confounded.

A curious pedunculated cyst sometimes sprouts from the labium minus. In 1881, Dr. Wiltshire exhibited before the Obstetrical Society two such cysts, removed from different patients. They contained translucent fluid. "They were attached in a slightly pendulous manner to the apex of the labia majora [*sic*, 'minora']

was evidently intended] and, as would be seen, were respectively the size of a small walnut and a hazel-nut. Such growths were preferably removed by the clamp and cautery." A similar specimen is preserved in the museum of St. Bartholomew's Hospital (3635A).

In 1888, Dr. R. T. Smith brought before the British Gynecological Society two cysts which he had removed from the labia minora of a woman aged 28, who had given birth to a child eleven years previously. There was no history of local injury.\* The cysts were within a third of an inch of the clitoris. It is certainly an interesting point in this case that the disease was bilateral. Mr. Bland Sutton observed that the cysts occupied a situation where dermoid sebaceous tumours might be met with; but they were of the mucous type.

I recently noted in a woman, aged 48, a symmetrical pair of obstructed sebaceous follicles, feeling as hard as dried peas, one in each labium majus, near the clitoris.

Hildebrandt dwells on vulvar cysts not developed from the vulvo-vaginal gland, but without any clear pathological definitions. He says that they are rare and vary greatly in size and in the thickness of their walls, which are closely connected with the neighbouring tissues. They are always, he states, unilocular, whilst amongst cysts which arise from the degeneration of Cowper's glands some are multilocular. On the other hand, they are sometimes multiple.

Judging from the cases of Wiltshire and R. T. Smith, excision is the right treatment for these cysts, and the proceeding offers no difficulty. Hildebrandt states that puncture and incision seldom cure; the surgeon should, therefore, endeavour to dissect out the cyst-wall. Here is one of the discrepancies of which I have already spoken. A pedunculated cyst does not require dissection. The operation, Hildebrandt remarks, is difficult, so that excision of a large piece of the wall, and plugging with lint soaked in iodine is sometimes the most complete operation that can be undertaken. This authority, it will be seen, discountenances the excision of cysts of the vulvo-vaginal glands.

The first important question in respect to cysts of the vulvo-vaginal glands is: do they arise, as a rule, from the duct or from the gland? If they arose from the duct alone, it would not be

\* Schröder attempts to trace these cases to injury.



difficult to dissect them away; but the pathology of these cysts must be considered before the necessary surgical treatment.

Huguier's classical monograph is not obsolete. It contains an excellent description of the vulvo-vaginal glands and their cysts. He states very clearly that cysts usually arise from one or more of the acini of the gland and are often entirely unconnected with the duct.

Dr. Stéphane Bonnet has composed the best of the more recent essays on the subject. He says that the cystic change is usually limited to the duct. He refers to Dr. Matthews Duncan, who doubted whether cysts of the gland ever occurred, but adds "ils existent, cependant." Indeed Dr. Bonnet proceeds to draw distinctions between duct-cysts and gland-cysts. A cyst of the duct is small and scarcely exceeds the size of a walnut, being usually no bigger than a filbert. It lies at the base of the lesser labium, which it unfolds. It is at first fusiform and becomes spherical. It is at first transparent, and a fine probe may be passed, in some instances, into the orifice of the duct, or some of the contents may be squeezed out.

A cyst of the gland, according to Dr. Bonnet, can at first be grasped. Then it lies behind the labium majus, between the vagina and ascending ramus of the ischium. It is spherical from the first. It may attain a large size and push forward both the greater and lesser labia. It is often not transparent, and the contents are frequently too thick to allow of evacuation through the duct by pressure.

Dr. Matthews Duncan's opinion has just been noticed. In his lecture "On Tumours and Cysts of the Vagina and Pudendum," he speaks of "the most common retention-cysts from closing of the aperture of one or both Cowper's ducts." In reference to cysts of the glands, he says: "I have never seen one, and I feel uncertain of the accuracy of the diagnosis in cases of which I have read, because they are described as deforming the labium and projecting into the vulva, and I should not expect this result to take place. If one of the glands of Cowper were enlarged by becoming diseased and forming a cyst, I should expect it to appear between the posterior extremity of a labium and the nearer tuber ischii, where tumours are produced by inflammation and abscesses of these glands, not in a labium." On the other hand, as this passage infers, Dr. Matthews Duncan believes in abscess of the



gland, and in his lecture "On Inflammations of the Pudendum," he describes this affection very carefully.

Dr. Auvar'd states that the gland itself may be transformed into a cyst by occlusion of its duct. Dr. Pozzi "ignores completely" any distinction between duct-cysts and gland-cysts; this opinion is the opposite to Bonnet's. I find the same division of opinion amongst German authorities. Winckel states that in cysts of the excretory ducts the labia appear as though divided into an inferior and superior portion. In cysts of the gland itself the distension is more in the lower and posterior portions.

From the great depth of the posterior part of the cyst in the case in my own practice, already described, I believe that it involved the gland. It corresponded to Bonnet's gland-cyst and occupied posteriorly the position in which Matthews Duncan would expect such a cyst to appear. I do not see why it might not have originated in the duct and gradually spread to the gland. The distension of the labia indicated cystic dilatation of the duct.

The cause of cystic disease of the vulvo-vaginal gland has been much disputed. There can be little doubt that the cyst is of the "retention" variety. On the retention theory, so probable in this case, it is quite easy to conceive that the gland itself may be involved in the cystic change. On the other hand, obstruction of the duct both at the point where it joins the gland and at its orifice, simultaneously, might, we believe, easily occur and set up a pure duct-cyst. I have never heard of a cyst of the vulvo-vaginal gland bearing the characters of a cystoma with solid contents. Hoening's case, which extended high into the pelvis, was possibly of Wolffian origin; no true cyst of the vulvo-vaginal gland ever invades the pelvic cavity.\*

If these tumours be retention-cysts, whence comes the retention? This question is not difficult to answer. Irritation of the orifice or canal of the duct causes its mucous lining to swell, and thus partial or complete obstruction may follow. Dirt, smegma, discharges, cicatrices (Goodell),† or bruises may constitute the

\* Dr. V. Chalot, of Toulouse, has written an important paper, entitled "Les Kystes Wolffiens du Vagin" (*Annales de Gynéc. et d'Obstet.*, July, 1892), in which he lays stress on the fact of a cyst extending to the lateral fornix, or further outwards. It seems to him conclusive evidence that the cyst is Wolffian.

† In Goodell's case the left duct was occluded after repair of a lacerated perineum.

source of irritation. Some authorities (Pozzi, &c.) insist that the irritation is necessarily gonorrhœal. This statement must be an exaggeration. Retention-cysts in other parts of the body are not rare, and at the same time have nothing to do with gonorrhœa. On the other hand, the exaggeration is natural. Undoubtedly inflammation of the duct is extremely common in gonorrhœa, and liable to persist after the remainder of the genito-urinary tract has recovered. In such a case the chance that a cyst may develop is considerable. The cyst must be primary, rather than the result of abscess of the gland, though there may be exceptional cases where the pus dries up and the abscess cavity gradually acquires the characters of a thin-walled cyst. A similar change occurs in the Fallopian tubes whereby a "pyosalpinx," that is to say, a much thickened tube containing pus, develops into a "hydrosalpinx," a large thin-walled cyst containing a clear serous fluid. A chronic abscess of the gland is most probably gonorrhœal in origin.

Dr. Stéphane Bonnet maintains that retention is no doubt the determining cause of cysts of the vulvo-vaginal glands, but not the sole cause. In many cases, he observes, the duct is open or the contents may at least be easily pressed out. This condition is, however, seen in retention-cysts elsewhere; a duct patulous at its orifice may be obstructed further back, and the obstruction may yield to external pressure. Dr. Bonnet suspects that an unknown degenerative process may take place in the glandular tissue. The change, he says, is sometimes associated with the development of cysts in the genital tract elsewhere. I have never found a vulvo-vaginal cyst in a case of ovarian cyst. In one case of cystic disease of the ovary, I detected a vaginal cyst, which bore no relation to the vulvo-vaginal glands.

Cysts of the vulvo-vaginal glands are believed to be most frequent on the left side, probably because too many writers of text-books have copied from Huguier's work. Out of 34 cases in Huguier's statistics, the cysts were unilateral in 29. 18 lay on the left side, 11 on the right. In 5 cases both glands were involved. Winckel, who sums up his own cases, says that he has observed the cysts 5 times on the left side, 6 times on the right, and in 1 case on both sides. We must now consider the question of treatment. Cysts of the vulvo-vaginal glands are treated by palliative and by radical measures. As a rule, the latter are the more satisfactory.

The palliative measures are catheterism and evacuation, drainage, seton, elastic ligature, incision, and apposition of the surfaces of the cyst wall, caustics, and excision of a piece of the cyst-wall.

Catheterism and evacuation may be followed by abscess; and so may puncture and incision. Both methods usually fail to cure.

Many years ago I applied a seton consisting of a stout piece of ligature-silk, to a large vulvo-vaginal cyst. Acute inflammation and suppuration followed; as there was free escape for the discharge, the patient suffered little pain. The cyst never refilled. I do not, however, advocate this practice. Drainage and elastic ligature are likewise not to be recommended. Incision, with subsequent apposition of the surfaces of the cyst-wall, however carefully done, seldom avails against recurrence.

Caustics, whether applied in the form of injections of strong solutions, or placed on lint as a dressing after excision of the cyst-wall, or applied direct to the remainder of the wall after partial excision, act by provoking inflammation, which causes the obliteration or destruction of the sac.

Excision of a piece of the cyst-wall, with the corresponding integuments, is recommended by Matthews Duncan and Schröder. Goodell advises the cauterisation of the remaining portion of the cyst-wall. Excision is the favourite method of those who object to extirpation. I have tried excision with cauterisation, after Goodell's method, but in one of my cases a fistulous tract remained, and proved hard to cure. I entirely agree with Bonnet in objecting to the employment of caustics in any form. The inflammatory process which they excite is hard to limit and much pain is inevitable; fistulæ are also very common after-complications.

The radical methods are destruction of the gland by the thermo-cautery and extirpation.

Although I have advocated the thermo-cautery, I do not at present use it, except as an auxiliary in case of hæmorrhage. When part of the cyst cannot be removed the thermo-cautery is the best agent for the destruction of the portion left behind. The same instrument is excellent in the treatment of old abscesses of the gland.

Extirpation of the entire cyst by careful dissection, as in the



case recorded at the beginning of this paper, is, I believe, the proper treatment in all cases of vulvo-vaginal cysts.

I admit that many authorities object to extirpation. Thus Goodell declares that "dissecting the sac out is a bloody operation and fortunately is rarely needful." He rightly observes that "fragments left behind are liable to reproduce the disease," but this objection applies to incomplete operations on any kind of cyst. Gaillard Thomas refers to wounding of the transversalis perinei artery and says that he has never found extirpation necessary. Hildebrandt also discountenances the radical operation on account of the danger of arterial hæmorrhage. In the case of very large cysts, damage to the rectum or even peritoneum may, he believes, occur. He apparently confounds vulvo-vaginal tumours with cysts of the Wolffian duct. This subject has already been noted in reference to Hoening's case and Chalot's monograph. The importance to the surgeon of a knowledge of the pathology of cysts of the lower part of the genital tract is here very manifest. Hildebrandt relies on excision of the walls of the cyst and plugging with lint soaked in tincture of iodine.

On the other hand, many writers of authority, or at least well-experienced, advocate, or even insist upon, incision. Stéphane Bonnet, who is in agreement with Trélat, holds that the gland must always be removed, because then alone can we be sure that the cyst will not recur. The incision, made in the line between the labium majus and minus, should exceed the diameter of the tumour. The process of dissection requires much care, patience, and time. The cyst wall "not rarely adheres to the bulb; hence double care is needed when dissecting near its level to avoid, as much as possible, damage to that structure." A hot antiseptic solution should be allowed to trickle over the field of operation; this will facilitate dissection and arrest hæmorrhage. As there are large venous plexuses near the cyst, rigorous antisepsis is necessary. The long wound must be closed with silkworm-gut and a drain left in the lower angle for a day or two. An iodoform-gauze tampon should be passed into the vagina and antiseptic dressing applied to the vulva. Hæmorrhage from the bulb is to be arrested by suture and the pressure of a pad. Torsion or ligature will secure a divided transversalis perinei artery. Bonnet's directions are well worthy of consideration.

Auvard thinks that puncture is insufficient. He prefers local to general anæsthesia when excision is undertaken.

Pozzi states that extirpation is preferable to any other treatment, as the least laceration of the cyst may render dissection tedious; he adopts an ingenious device to prevent, as far as possible, that accident, or to render it as little troublesome as possible should it occur. He punctures the cyst with a hydrocele-trocar, lets out the contents, washes out the cavity with hot water, and injects spermaceti warmed at a low temperature in a water-bath. Ice is then applied and in a few minutes the cyst forms a hard mass which may be quickly removed. The anæsthesia caused by the ice, aided at the most by cocaine, is sufficient, without chloroform.

J. Levrat believes that removal of a vulvo-vaginal cyst is the only operation that thoroughly prevents recurrence. "This appears to us all the more important, inasmuch as we believe, on the strength of the two cases where we operated and examined the growths, that certain cysts may be the first stage of yet more serious tumours." An important statement, but I have seen and heard of numerous cases of incomplete operations, some very carelessly performed and highly unsuccessful, yet I know of no instance of subsequent malignant changes.\*

I have heard of one case where fatal hæmorrhage followed excision, but there was evidently want of care in after-treatment. My own case was severe owing to the size of the cyst and to deep adhesions and vascular connections, yet the cyst was thoroughly extirpated. On that account I recommend extirpation even when the cyst is large. The disease which we have been considering is a nuisance to the patient, and that is why she seeks relief. A fistulous tract remaining after a "minor" operation is a greater nuisance than the cyst itself. Hence the operation which gets rid of the cyst and leaves no fistula is the best.

#### REFERENCES.

AUVARD. 'Traité Pratique de Gynécologie,' 1892, p. 169.

BONNET (Stéphane). "Des kystes et abcès des glandes vulvo-vaginales." 'Gazette des Hôpitaux,' 1888, p. 637.

---

\* Since this memoir was read before the Society, an important paper on "Carcinoma glandulæ Bartholini," by Dr. F. Schweizer, has appeared in the 'Archiv für Gynäkologie,' vol. xlv, Pt. 2. It justifies Levrat's opinions above noted, and is a further argument in favour of extirpation of cysts of the gland.

- DUNCAN (Dr. J. Matthews). 'Clinical Lectures on the Diseases of Women,' 3rd ed., 1886, pp. 172, 191.
- GALABIN. 'Trans. Obstet. Soc.,' vol. xxvi, 1884, p. 56.
- GOODELL. 'Lessons in Gynæcology,' 3rd ed., 1887, p. 88.
- HILDEBRANDT. 'Die Krankheiten der aeusseren weiblichen Genitalien' 1877, pp. 56 and 66.
- HOENING. 'Monatschrift f. Geburtshülfe,' vol. xxxiv, p. 130.
- HUGUIER. "Mémoires sur les maladies des appareils sécréteurs des organes génitaux externes de la femme." 'Mémoires de l'Académie Nationale de Médecine,' vol. xv, 1850, pp. 527—847.
- LEVRAT. Article "Vagin et Vulve." 'Nouveau Dictionnaire de Médecine et de Chirurgie Pratiques,' vol. xxxviii.
- POZZI. 'Traité de Gynécologie,' 1890, p. 1028.
- SMITH (R. T.). 'British Gynæcological Journal,' vol. iv, 1888, p. 6.
- THOMAS (Gaillard) and MUNDÉ. 'Practical Treatise on Diseases of Women,' p. 157.
- TRÉLAT. Lecture, 'Gazette des Hôpitaux,' 1887, p. 910.
- WILTSHIRE. "Cysts from the Labia Minora." 'Trans. Obstet. Soc.,' vol. xxiii, 1881, p. 206.
- WINCKEL. 'A Handbook of Diseases of Women' (translated by Dr. T. Parvin), 2nd ed., 1890.

*November 7th, 1892.*

## TUBAL MOLES AND TUBAL ABORTIONS.

By J. BLAND SUTTON, F.R.C.S.

IN a paper read before the Roy. Med.-Chir. Soc., November 12th, 1889, I drew attention to the fact that the ovum in a case of tubal pregnancy is liable, like the ovum in uterine pregnancy, to become converted into a mole (apoplectic ovum). Since that date my observation has been abundantly confirmed in Germany, America, and England; indeed, the condition is now so well recognised that it seems almost superfluous for me to again direct attention to it. However, I feel it necessary to do so on account of the many opportunities I have enjoyed of studying the tubal mole since the original paper was published; also it is necessary to correct a few misconceptions.

Tubal moles differ from uterine moles in several particulars; indeed the points of distinction are such as to enable us readily to tell one from the other. The uterine mole is more or less spherical; the amniotic cavity is of fair size, and occupies the centre of the mole. The embryo may or may not be present.



Sometimes it is represented merely by an ill-shaped mass pendulous at the end of the cord. Even when the embryo can be recognised it is very misshapen and the umbilical cord is often cedematous (Fig. 1). A tubal mole in its early stage is spherical,

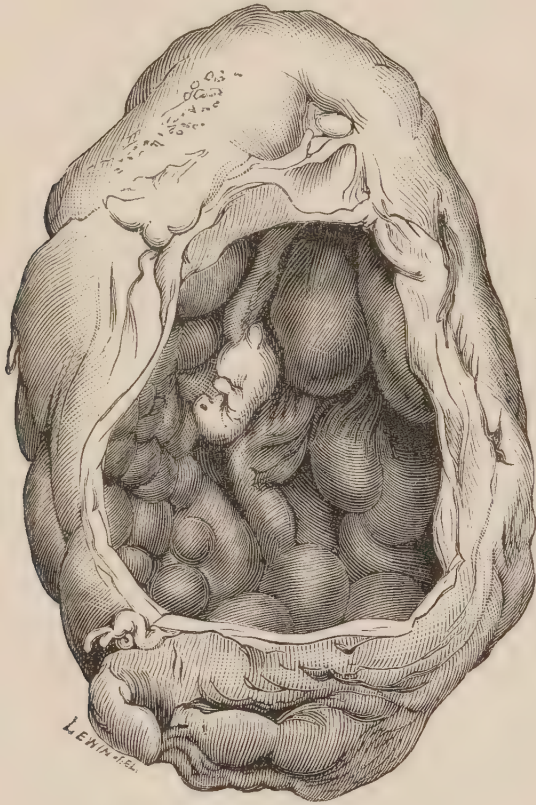


FIG. 1.—A typical uterine mole.

but, after attaining the dimensions of a walnut, becomes ovoid (Fig 2). In the majority of cases the amniotic cavity occupies an excentric position; in consequence of this peculiarity the thin amnion is easily ruptured, and permits the escape of the embryo. This explains the difficulty of finding the embryo in many cases where the mole has been discharged through a rent in the wall of the tube or aborted through an unclosed ostium, accompanied, as the rule is in these cases, with free hæmorrhage. The mole is easily found in the clot, but if the embryo has escaped from the amniotic cavity the chances are that it will not be recognised. It must not be imagined that because the embryo has not been found it has been dissolved by the peritoneum. On one occasion I collected all the blood and clot which I removed during an operation

five weeks after the rupture of a gravid tube, and disintegrated it by a gentle stream of water. In the course of this manoeuvre the embryo came to the surface, was promptly recognised, and caught. As is the case with uterine moles, the embryo sometimes dies very early, and the amniotic sac contains nothing but a small quantity of fluid. When the mole, on its escape from the tube, is discharged between the layers of the broad ligament it becomes so compressed that the embryo is found flattened out like a succulent flower firmly squeezed between the leaves of a heavy book.

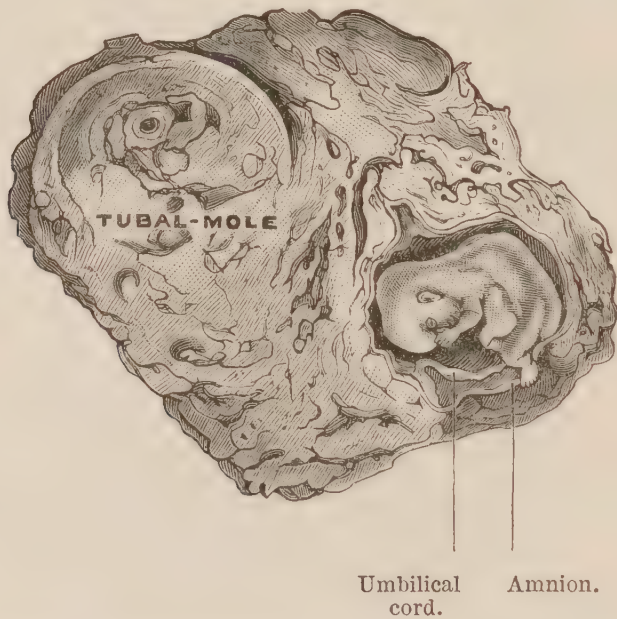


FIG. 2.—A tubal mole in longitudinal section. (From a specimen in the possession of Dr. Walter.)

There is rarely any difficulty in recognising a tubal mole. When an embryo is present there is no room for doubt. This is equally true when the mole contains an amniotic sac, though no embryo be present (Fig. 4).

In hard firm clots in which the amniotic cavity is not recognisable, sections from the supposed mole must be prepared and examined with a microscope for chorionic villi. The presence of chorionic villi are as indicative of a mole as the presence of an embryo. These villi are such characteristic structures that they cannot be confounded with "half-organised blood-clot," as some writers have suggested. The use of such an expression implies ignorance of even the elementary facts of histology. When seen in



stained sections among blood-clot the villi are very striking objects, and in order to facilitate their recognition Fig. 3 has been prepared. They usually appear in sections as clusters of circular bodies; ten or more in fortunate sections may be counted together. More frequently they occur in groups of three or four, and often a wide section of clot will be examined without finding more than two or three. Under a low power they present an external layer of

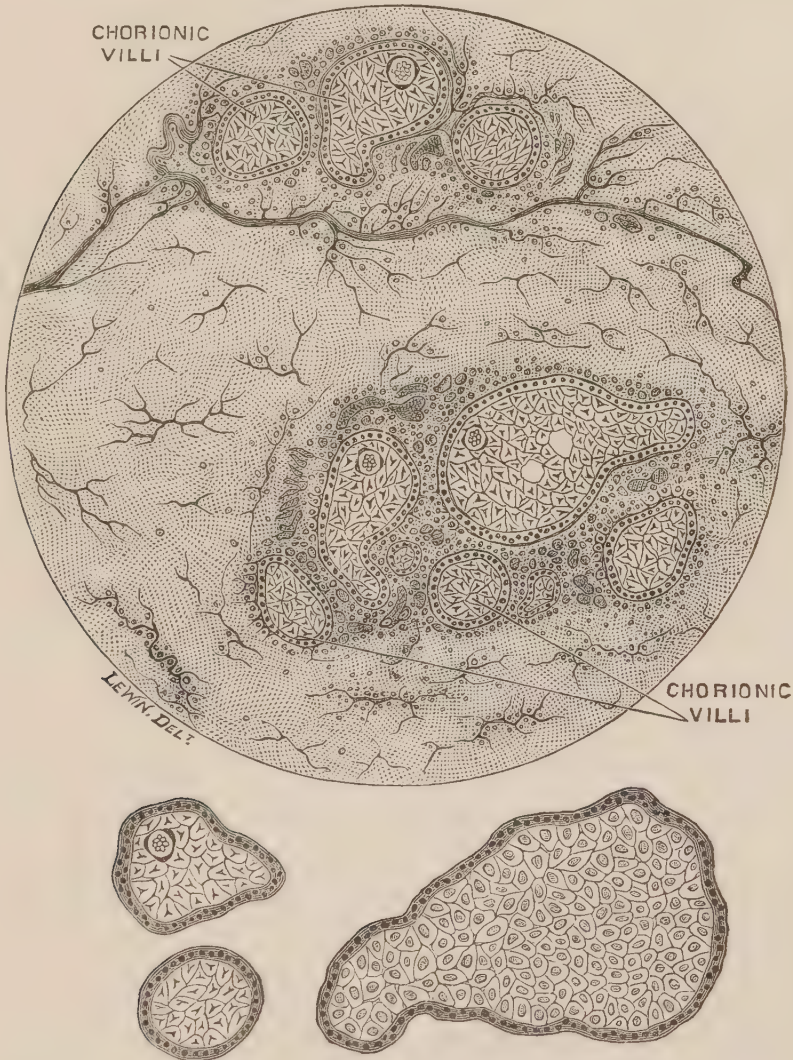


FIG. 3.—Chorionic villi from a tubal mole (magnified) embedded in blood-clot. B, Villi highly magnified.

epithelial-like cells, the central space being occupied by cells of irregular shapes. When examined under a high power the limiting layer is seen to be formed of a perfectly regular row of cubical epithelium; sometimes two rows of epithelium are present.

It is of great importance to appreciate clearly the characters of



a tubal mole, for the presence of a mole is decisive proof of pregnancy. It must not be supposed that every blood-clot found in a Fallopian tube is a mole. That blood and blood-clots are occasionally found in the Fallopian tube unconnected with pregnancy is beyond all dispute. Anyone who has taken the smallest interest in the history of any branch of medicine must be aware that originally, many conditions which we now know to be distinct were formerly grouped together under a comprehensive term. It was customary to apply the term "hæmatosalpinx" to accumulations of blood in the Fallopian tubes independently of their origin. The discovery of the tubal mole has furnished a criterion for the differentiation of gravid tubes. Formerly the majority of Fallopian tubes containing blood-clot were classed as examples of hæmatosalpinx, and an examination of museum specimens demonstrates this absolutely. For the sake of accuracy in the future it will be necessary to reserve the term "hæmatosalpinx" for a non-gravid Fallopian tube distended with blood secondary to occlusion of the abdominal ostium.

The consideration of the occlusion of the abdominal ostium of the tube brings me to the question of tubal abortion. The retention of an impregnated ovum in the Fallopian tube leads to occlusion of the abdominal ostium, an event usually complete by the sixth, but often delayed to the eighth, week following impregnation. It is therefore a comparatively slow process. (When the ovum is lodged in the ampulla of the tube the ostium cannot close.) So long as the tubal ostium remains open the ovum is in constant jeopardy of being extruded through it into the peritoneal cavity, especially when the ovum lies near, or in, the ampulla of the tube. When an impregnated ovum is thus extruded from the tube into the general peritoneal cavity it is invariably in the condition of a mole, and the accident is always accompanied by hæmorrhage. The extrusion of a mole in this way is indicated by the term "tubal abortion." Free hæmorrhage may occur from a gravid tube and the mole be still retained in consequence of its attachment to the wall of the tube. Under such conditions the bleeding may be repeated; this is known as "incomplete tubal abortion." Some of the most striking instances of recurrent hæmorrhages from gravid tubes occur when the ovum is too large to pass through the ostium. There is a variety of dilated tube which resembles a wine jar (amphora) without handles, and the

ovum, though free to move about within the dilated portion of the tube, is as safely imprisoned as the wooden pea in a schoolboy's whistle. In many of the specimens the mole remains united by a portion of its circumference to the tubal mucous membrane.

A gravid tube may rupture and the mole slip into the peritoneal cavity through the unclosed ostium. The amount of blood which may escape in these cases is truly astonishing, as the following case will serve to show.

Mary B—, 27 years of age, married, mother of one child. She had menstruated regularly for five years. On June 20th, 1892, she was poorly as usual. July 20th, she had a slight brown discharge. August 2nd, the patient, when out walking, was suddenly seized with severe cramp in the belly. August 4th, she was ill with abdominal pain and vomiting. The patient was under the care of Dr. J. J. Clarke, of Walthamstow, and on August 8th he found her collapsed, pale, pulse scarcely perceptible at wrist, sighing respiration, and the voice weak. I was summoned, and found her in the condition described above. Dr. Wise, of Walthamstow, was also present, and it was clear that the patient was suffering from severe internal hæmorrhage. Abdominal section was performed without delay. On incising the parietes an enormous quantity of blood gushed out. The right uterine appendages were drawn out through the wound and the mole depicted in Fig. 4 was found among the fringes of the tube. The broad ligament was transfixed, tied, and the tube with the ovary cut away. The left ovary and tube appeared healthy and were, therefore, not interfered with. The effused blood was flushed out with warm water and the abdomen closed in the usual manner. On returning the patient to bed no pulse could be felt at the wrist and she was very cold. With the aid of hot bottles, brandy enemata, and small quantities of brandy by the mouth, the signs of collapse slowly passed off, and in fourteen days the patient was convalescent.

The parts removed are depicted of natural size in Fig. 4. The ampulla of the tube is widely dilated and there is a small rent in its wall.

When a Fallopian tube becomes gravid a decidua forms in the uterus, and this curious structure has a diagnostic value. The decidua is rarely retained until the completion of gestation, and thrown off during the false labour. Usually it is discharged during the early period of pregnancy, often in fragments, unaccompanied by pain, or it is expelled whole with symptoms of miscarriage. When a decidua is discharged whole it represents a cast of the uterine cavity. It is in shape pyramidal; the base corresponds to the fundus of the uterus, and at each angle there is an orifice corresponding to the uterine opening of each Fallopian



tube. The apex of the pyramid is occupied by a circular hole corresponding with the dilated internal orifice of the cervical canal. This is smooth and rounded, whilst the smaller orifices

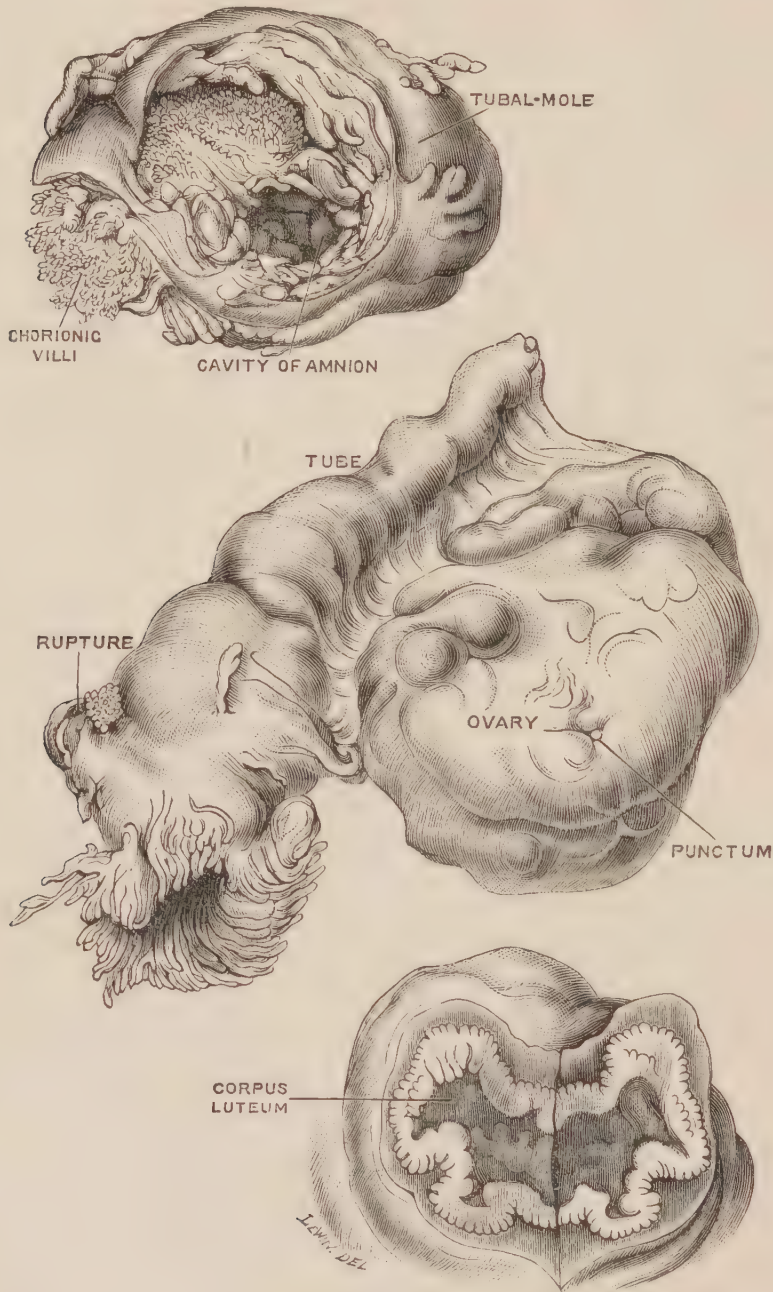


FIG. 4.—A gravid tube in which rupture occurred. The mole escaped through the unclosed ostium, and at the time of operation was lying among the fimbriæ.

corresponding to the uterine ends of the tubes are ragged. The exterior of the decidua is shaggy, the interior smooth and dotted



with many minute puncta representing the orifices of the uterine glands. (Fig. 5.)

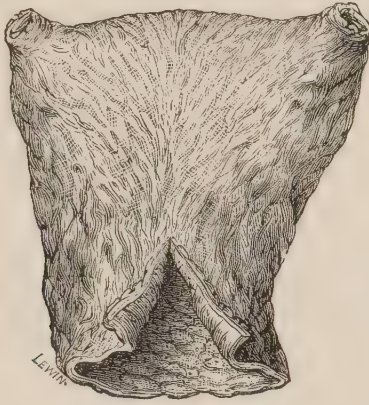


FIG. 5.—A uterine decidua formed in a case of tubal pregnancy, discharged at the end of the eighteenth week of gestation.

The unnecessary formation of a decidua in the uterus when a tube is gravid, or in the cornu of a bicornuate uterus when the opposite cornu is impregnated, is a curious illustration of the traditional way Nature does her work, and teaches a lesson some of us may well take to heart. There is little doubt that the majority of cases formerly classed as pelvic hæmatoceles are, in reality, the result of rupture or abortion of gravid tubes. The cases in which doubts arise are those in which women are seized with symptoms indicating internal hæmorrhage; on opening the abdomen free blood is found, often in abundance; the tube is widely dilated, perhaps ruptured, and clots of blood hang about the fringes, yet no embryo or mole is detected. In some of these cases a decidua is discharged from the uterus. These doubtful cases will become fewer as operators become familiar with the tubal mole. The mole is so different from ordinary clot that there should be little difficulty in distinguishing it, except in cases in which it is very small.

The following is a summary of the views contained in this paper:—

1. The transformation of a tubal ovum into a mole is beyond doubt.
2. The majority of specimens described as examples of hæmato-salpinx are gravid tubes.

3. Rupture of a gravid tube and tubal abortion are the common causes of pelvic hæmatocele.
4. Mesometric rupture of a gravid tube is a common cause of pelvic hæmatoma.
5. Every clot of blood found in a Fallopian tube is not a tubal mole.

Since the discovery of the tubal mole, specimens of occluded Fallopian tubes filled with blood, independent of tubal pregnancy, are now found to be infrequent. In the last report of the Museum of the Royal College of Surgeons (1892), I notice a description of "An Unequivocal Example of Hæmatosalpinx." This is a fair indication of the revolution which has taken place in our knowledge of the early stages of tubal pregnancy.

Dr. CULLINGWORTH said that the obstetric physicians were always ready to recognise the excellent work done by Mr. Sutton in connection with their department, notwithstanding the hard things Mr. Sutton from time to time said of them. He happened to be present at the meeting of the Royal Medical and Chirurgical Society in 1889 when Mr. Sutton read the paper to which he had referred, on tubal moles, and he remembered supporting Mr. Sutton that evening in his contention that no case should be described as a tubal gestation unless undoubted evidence was forthcoming of the presence of foetal structures, either embryo, or chorionic villi, or amniotic sac. After what had fallen from Mr. Sutton, and after an increased clinical experience of his own, he was not now prepared to take up quite so strong a position. The products of conception might be exceedingly minute and, especially if the ovum had been expelled into the peritoneal cavity, might be lost in the midst of a mass of blood-clot and escape detection. So that he did not agree with Mr. Sutton in thinking that the time had come for restricting the word hæmatosalpinx to accumulations of blood in the Fallopian tube from causes other than tubal gestation. It would be wise to retain the name as denoting a Fallopian tube distended with blood from whatever cause. Until we could say with absolute certainty this is a case of tubal gestation whilst that is not, it would be better to retain a comprehensive term, which would cover both, and then give the varieties. If we define too minutely we shall go beyond our knowledge, and find ourselves in difficulties. He would like to ask Mr. Sutton if he was of opinion that, after the escape of an ovum through the abdominal ostium of the tube, the ostium might undergo further changes, and, becoming occluded, might close in upon the blood remaining in the tube.

Dr. PETER HORROCKS said that whilst a typical chorionic villus was easily distinguished from a typical blood-clot when both were submitted to microscopical examination, he confessed himself unable, sometimes, to be quite positive in atypical specimens, where alterations, *ante* or *post mortem* or both, had so changed the appearances as to quite spoil the usual characteristics. He mentioned a case in his own practice where he had with great difficulty and after making many different sections, come to the conclusion that it was really a product of gestation. He disagreed



entirely with the proposal that the term hæmatosalpinx should be limited to those cases where blood was found in the tube owing to some cause other than tubal gestation. He also objected to limiting it to cases where the blood was clotted and the tube sealed. He considered it best to define hæmatosalpinx as "blood in the Fallopian tube," irrespective of its origin or its condition, fluid or solid, liquid or clotted, or of the condition of the tube itself or its ostia. Blood in the tube, wherever it came from, was abnormal and so pathological. It would be most confusing to students if they were taught that hæmatosalpinx meant blood in the tube only when due to a particular cause. The different causes could easily be differentiated subsequently, but he could see no advantage whatever in the proposal made to limit its meaning. He asked what was the source of the profuse hæmorrhage in the cases mentioned by Mr. Bland Sutton, also, was it possible for chorionic villi to grow after the death of the embryo. He had reasons to believe this possible.

Dr. LEWERS wished to say a word or two as to the natural history of these cases of extra-uterine gestation. He believed that in many cases where the foetus died at an early period, complete recovery often took place without operation. Even when rupture had occurred, giving rise to pelvic hæmatocele, many of the cases did quite well under expectant treatment. He referred to a case of pelvic hæmatocele, due to rupture of an extra-uterine foetation, that had been under his care at the London Hospital some six years ago, and where he removed the foetus through an incision into the posterior vaginal wall. This patient made a complete recovery, but he believed she would have recovered equally well without interference. At the same time he quite recognised that there was a class of cases requiring abdominal section, but it was often a difficult matter to distinguish between such cases and those that would do as well without it. Evidence of the continued development of the gestation sac furnished a valuable indication.

Dr. HEYWOOD SMITH inquired of Mr. Sutton the exact source of the hæmorrhage when large in amount. He supposed that the blood was usually poured out before the ovum reached the end of the tube. Did the blood come from the ruptured attachment of the ovum or from the ruptured tube? He asked, also, as to the usual locality of adhesion of the ovum to the tube. He likewise desired to know in what cases one might expect tubal abortion and whether there was anything usually to account for it, such as an accident, a slip, a jerk, or the passing of hard fæces, dislodging the ovum.

Dr. ROUTH quite believed that in most cases hæmatosalpinx, or tube moles, were the result of ectopic gestation. But he thought it was not always wise to trust only to the pathological appearances; if you found remains of the foetus or chorionic villi well and good. But if not, it was not prudent to say it was necessarily not an ectopic gestation. He was surprised that the obstetricians present had not spoken of the preceding symptoms of pregnancy which must have existed in any ectopic gestation, especially (and one case cited was a nine weeks' pregnancy) the escape of decidua from the uterus, either whole or partial. These would help the diagnosis. He cited, however, one case of true hæmatosalpinx in confirmation. A girl had retention of the menses, from absence of an os uteri. The uterus was reaching to the umbilicus. He punctured the uterus through the vagina, and subsequently injected the cavity, when voided, with iodine, the usual treacly fluid having escaped. She did very well for about four days, and then he, Dr. Routh, was sent for suddenly.



The girl had been sick, had felt something give way inside, was blanched and collapsed, &c. All the symptoms of a ruptured ectopic gestation were present. In those days he was not allowed to open the abdomen. She died. The *post-mortem* revealed a lengthened tube on the left side with two dilatations, one the size of a large walnut, the other somewhat smaller. Both ends of the tube were closed, nor was there any communication with the two dilatations, the tubes being impervious throughout. It was the larger dilatation that had given way and caused death, much blood having been lost. On the right side there was a smaller dilatation also. Here were marked symptoms of ruptured ectopic gestation and yet only tubal hæmatocele, for she was a pure virgin and about 16 years old only.

Dr. W. S. A. GRIFFITH, while criticising some of the details of the paper, did so with the full recognition of the value of Mr. Sutton's contributions to this subject, but ventured to think that pathologists were indebted to him for the name "tubal mole" rather than for their knowledge of its occurrence. He agreed with those who objected to the proposed limitation to the term hæmatosalpinx, and whatever might be said against the abuse of definitions, their proper use was of the highest value. There was one form to which Mr. Sutton had not alluded, namely, that in which the tubal mucous membrane bled, apparently in connection with and at the same time as the uterine mucous membrane. Dr. Griffith had described these cases and demonstrated it in two specimens, and was of opinion that regurgitation from the uterine cavity, although supported by the opinion of Dr. Matthews Duncan, was exceedingly rare, if, indeed, it ever occurred, except as a result of atresia of the cervix, and in these cases there were special characters which distinguished them, the retained blood undergoing decomposition, and so having the character of blood retained in the vagina. Drs. Ballantyne and Williams, in their investigations into the anatomy of the tube, had been able to demonstrate the mechanism of the closure of the fimbriated end more completely than Mr. Sutton had done. They showed that the ring referred to by him was the limit of the circular muscular fibres, while the longitudinal fibres were continued to the extremities of the principal fimbriæ; the obvious inference being that the fimbriæ were drawn in under certain conditions by these fibres. Dr. Griffith objected to the common expression "rupture" of the tube into the broad ligament, on the ground that under ordinary circumstances the process was one of invasion, passive and gradual, not sudden and instantaneous. Mr. Sutton was much given to criticising others for inaccuracies of fact and statement, and often very deservedly so, but he was by no means free from similar mistakes himself. He had, in the course of the evening, drawn inferences which were of some importance if they were well founded, but the evidence on which he relied was founded on clinical and other distinctions between pregnancies of the fourth, fifth, sixth, and seventh weeks. He would ask Mr. Sutton how he managed to obtain reliable distinctions.

Mr. SUTTON, in reply, said that the main object of his paper was to give a careful account of these specimens so that others might be familiar with their external characteristics. After abortion or rupture of the tube the closure of the ostium would be deferred. He thought that the case related by Dr. Lewers was probably a hæmatoma produced by rupture of the gestation sac between the layers of the broad ligament. The dates of pregnancy he had given were approximate and were reckoned from the last period. He had examined a specimen of a well-formed mole in a

tube without rupture, and this had led him to modify his former belief on this subject. After the embryo died he felt sure there was no continued growth of the villi.

---

*November 21st, 1892.*

## CONGENITAL SYPHILIS AS A CAUSE OF NERVOUS DISEASES IN CHILDREN.

By W. B. HADDEN, M.D. Lond., F.R.C.P.

SCANTY as are the records of morbid anatomy, there is, nevertheless, reason to believe that "nearly every variety of nervous affection of acquired syphilis has its parallel among congenital examples."\* But the question arises whether congenital syphilis is an important agent in the causation of such nervous disorders as infantile hemiplegia, cervical opisthotonos, and the clinical conditions which are due to sclerosis of the convolutions.

*Hemiplegia.*—Infantile hemiplegia is conveniently classified on clinical grounds into cases (*a*) having an acute onset, (*b*) having no definite onset. Many of the latter are, no doubt, due to meningeal hæmorrhage occurring during labour; but this cause cannot always be invoked. In fifteen cases of this class I found evidence of congenital syphilis in the family or personal history in four or five instances, without any history of difficult labour. Hemiplegia with acute onset is particularly common under the age of 3 years, and although a certain number follow the acute specific fevers, the greater proportion arise without apparent cause. Embolism at this period of life may be practically excluded. There is reason to think that the lesion is usually either thrombosis or hæmorrhage, so the inquiry resolves itself into the causation of arterial degeneration in young children. Patho-

\* "Syphilitic (Hereditary) Diseases of the Nervous System," by Dr. Barlow and Dr. Judson Bury, 'Dictionary of Psychological Medicine,' edited by Dr. Hack Tuke, vol. ii, p. 1259.



logical investigations on the condition of the vessels in inherited syphilis are not numerous, but sufficient evidence has accumulated to show that arterial disease is not so uncommon as was formerly supposed.\* I have lately found marked arterial changes in an infant, aged 6 weeks, in whom there was advanced cirrhosis of the liver, with unmistakable evidences of congenital syphilis.

There is considerable divergence of opinion among purely clinical observers as to the influence of congenital syphilis as a cause of infantile hemiplegia. Osler† ascribed only 1 case out of 120 to syphilis; but it must be remembered that many of his observations were made years after the attack, when the evidences of syphilis were not readily to be made out. Abercrombie, whose investigations were made soon after the attack, ascribed 4 at least to syphilis, probably 6, out of a series of 50 cases;‡ 2 cases only out of 83 analysed by Sachs and Peterson were ascribed to inherited syphilis.§ Marie, Fournier, Henoch, and Hutchinson evidently consider infantile hemiplegia as being rarely syphilitic. Clinical observations at hospitals devoted to children's diseases appear to indicate that syphilis may have a more important etiological share than is generally allowed. Out of 25 cases of hemiplegia with acute onset, I found no history bearing on syphilis in 9; in 3 instances special inquiries were not made on this point; in 5 there was a suspicion of syphilis, and in 6 the evidence was stronger; in 2 cases the children were certainly syphilitic.

CASE 1.—In a child, aged 9 years, there was ulceration of the palate and caries of the nasal bones, as well as iritis and keratitis. The patient had been taking mercury for three months, when epileptiform attacks occurred. At the end of six months, in spite of treatment by mercury, hemiplegia supervened, and the child subsequently became idiotic.

CASE 2.—A boy, aged 10 years; hemiplegia had occurred when he was 1 year and 9 months old. He had interstitial keratitis and extensive choroidal atrophy.

\* See article already quoted by Dr. Barlow and Dr. Judson Bury; also cases by Dr. Barlow, and by Drs. Turner and Sutton, in vol. xxviii, 'Trans. Path. Soc.'

† "The Cerebral Palsies of Young Children."

‡ "Clinical Lecture on Hemiplegia in Children," 'Brit. Med. Jour.,' June 18th, 1887.

§ "A Study of Cerebral Palsies of Early Life, based upon an Analysis of 140 Cases," 'Journal of Nervous and Mental Disease,' May, 1890.



CASE 3.—Syphilis was suspected in a child, aged 14 months, who had two distinct attacks of hemiplegia.

CASE 4.—In another case, an idiotic child, probably syphilitic, was attacked suddenly with hemiplegia.

CASE 5.—In another patient, hemiplegia occurred at the age of 7 years, and after the lapse of five years the limbs on the opposite side gradually became rigid, possibly from chronic meningitis or sclerosis of the convolutions.

*Cerebral Sclerosis.*—There is good evidence that sclerosis of the cerebral convolutions is sometimes due to inherited syphilis. In one case, in which there was slowly-increasing rigidity of the arms and legs, with mental impairment, in a child aged 2 years, I found marked sclerosis of the convolutions. In the notes it was simply stated that the child had suffered from snuffles and a rash on the buttocks in babyhood, but I found afterwards that the father had been under the treatment of one of my colleagues for syphilis before the birth of the child.

*Disseminated Sclerosis.*—Dr. Moncorvo has brought forward some evidence that disseminated sclerosis in the child may be syphilitic. In a case of this kind occurring in a boy, aged 8 years, a brother was brought to me with a typical syphilide. In two other cases of this disease I had reason to suspect syphilis.

*Idiocy.*—The etiology of idiocy is too large a question for discussion now, but I may briefly say that some non-congenital cases are indisputably of syphilitic origin. It is difficult, however, to determine the relative frequency of this cause. Sclerosis of the convolutions or extensive chronic meningitis is sometimes the underlying condition, and the mental defect may or may not be associated with rigidity of the extremities.

*Convulsions.*—It is important to bear in mind that convulsions may indicate actual structural changes in the brain. Infantile eclampsia may be almost the sole indication of syphilis affecting the higher nervous centres, and it is a clinical fact that seizures of this kind will sometimes yield to mercury, when bromide of potassium and all other remedies have failed.

*Cervical Opisthotonos.*—In this condition the head is strongly retracted, and attempts to put it forward give rise to pain. The fontanelle is often bulged, and the sutures may widen out. Hydrocephalus, indeed, often supervenes. Paralysis of the cranial nerves and optic neuritis are not generally present. The active

symptoms, with partial or even complete remissions, may last for months. Sometimes death ensues at a comparatively early period, sometimes the patient lives to be hydrocephalic, sometimes recovery takes place. This condition is due to simple (non-tuberculous) meningitis, chiefly at the base, more especially affecting the membranes between the medulla and cerebellum. Sometimes there is spinal meningitis, particularly on the posterior surface. Cervical opisthotonos is sometimes associated with clear evidences of congenital syphilis, and a causal connection may reasonably be assumed, at any rate in some cases. The frequent coincidence of the two states can scarcely be fortuitous. It is curious, however, that mercury will sometimes be of little use in such cases.

Dr. ALTHAUS expressed his regret at the absence of the President, Mr. Hutchinson, from the meeting, as he had done more than anyone else to facilitate the diagnosis of congenital syphilis in children, and as any remarks from him on the subject submitted to the Society would no doubt have been very valuable. In Dr. Althaus's experience of syphilitic hemiplegia in children, this affection was usually, if not invariably, accompanied by other symptoms pointing to the dyscrasia, such as scars, more especially about the lips, a peculiar malformation of the skull, a sallow complexion, imperfect development of the body altogether, intellectual torpor, sarcocele, &c., in addition to one or all of Hutchinson's symptoms. Syphilitic hemiplegia was often preceded by epilepsy, which at first sight might appear to be idiopathic, but was, sooner or later, by the cropping up of additional symptoms, revealed to be of specific origin. In acquired syphilis of infants, hemiplegia appeared to be much rarer than in the congenital form of the disease. A persevering use of combined treatment by mercury and large doses of iodide of potassium often gave very good results, while either of these agents employed singly was not nearly so useful.

Mr. SHEILD remarked that the deafness in syphilitic children was usually not due to otitis media, but a large majority of cases had complete nerve deafness caused by an effusion into the tissues of the labyrinth and auditory nerve.

Dr. LEES said that syphilis was, no doubt, a cause of disease to a much greater extent than it was possible actually to prove ; but, on the other hand, the question of coincidence must be considered ; and it did not follow that because syphilis existed it was the cause of the symptoms actually present. In the cases of infantile hemiplegia which he had seen, he had rarely had reason to believe that they were due to syphilis. The same was true with regard to posterior basic meningitis, of which he and Dr. Barlow had seen a large number of instances, nearly a hundred, with about thirty autopsies. No doubt some of these cases improved on mercury and iodide, but that could not be accepted as proof of their syphilitic origin. There were, however, some diseases of the nervous system in early life which were undoubtedly syphilitic. Gummata on the cranial nerves were met with occasionally, and he related a case of temporary paralysis of the 6th and 7th nerves which had recovered on



mercury and iodide, and may possibly have been of this nature, though he could not discover any evidence of syphilis. He also narrated a case of gumma of the dura mater in a girl of 13, with tenderness of skull and left-sided convulsions, at present under his care at St. Mary's Hospital. Sclerosis of the brain was also sometimes, unquestionably, due to syphilis, and an account was given of this condition in a boy recently under his care at the Hospital for Sick Children.

Dr. BARLOW said that he could recall about six cases of hemiplegia in congenital syphilis, in two of which necropsies were made. In one, a girl aged 9, who had been under observation for five years, there were all the signs of inherited syphilis; she had a convulsion, followed later by the development of hemiplegia, first on one side and then on the other; and she, later, passed into a state of hebetude. There was found extensive and typical syphilitic disease of all the arteries of the circle of Willis, with marked sclerosis of both hemispheres. He said that the rule was to get generalisation of the disease all over the nervous system—first convulsions, local or general, then hemiplegia, perhaps spasm on one side and palsy on the other; then choroiditis, interstitial keratitis, and, finally, idiocy. Such was the typical course of cerebro-spinal syphilis. The prognosis was always bad, though the symptoms might be relieved by iodide of potassium and mercury. The true basis of observation was morbid anatomy, and it should be remembered that every kind of lesion found in acquired syphilis was found also in the congenital affection, though the distribution was different.

Dr. WALTER CARR said, that a condition so common as congenital syphilis was bound to occur rather frequently along with other diseases as a mere matter of coincidence, without there being any necessary relation of cause and effect, and, therefore, the fact that in a small percentage of cases of nervous diseases in children there was evidence of congenital syphilis, was very far from being any proof that the latter could, in any way, be considered as a factor in the causation. Speaking with especial reference to posterior basic meningitis, he said that, after observing some half dozen cases, in none of which was there any evidence of syphilis, he was especially struck with the definite symptomatology of the disease, varying only in regard to acuteness or chronicity, and still more with the definite localisation of the pathological changes, notably with the invariable thickening of the pia mater between the cerebellum and medulla, and also at the tip of each temporo-sphenoidal lobe. He could not conceive that a condition so definite could be produced by several diverse causes, of which syphilis could certainly only be one occasionally; and he could only believe that there must be some one ætiological factor—as definite as for tubercular meningitis—which had up to the present evaded our observation.

Dr. WHEATON said that he was surprised that Dr. Hadden had come to the conclusion that hemiplegia in children was frequently due to syphilis. He thought that the presence of any fixed and unchanging paralysis, such as hemiplegia, was against the diagnosis of syphilis, the nervous symptoms due to which were generally of such a variable and changing character. It was possible that the cases recorded by Dr. Hadden, in which hemiplegia followed repeated attacks of localised convulsions, were examples of insular sclerosis affecting the cerebral cortex. The histories of these cases appeared to be typical of the latter disease, of which, however, syphilis might be an important factor in the causation. He mentioned a case in which a child, aged 3, was admitted into the hospital on



three separate occasions, suffering from unilateral convulsions followed by hemiplegia. The hemiplegia was temporary at first, but became permanent later. At the autopsy, hard patches of sclerosis, resembling pieces of cartilage, were found scattered over the convolutions on both sides of the brain. The presence of caseous nodules in the cerebral cortex might also produce similar symptoms. Children were sometimes admitted into hospital for surgical operations, in whom all signs of nerve disease were absent, and after recovery from the anæsthetic localised convulsions would occur, followed by paralysis. These convulsive attacks might recur again and again, and the patients might live for a long time afterwards. On *post-mortem* examination, hard, yellow nodules would be found in the cerebral cortex which could be proved to be tubercular on microscopical examination only. In many cases of syphilis in children, cerebral symptoms were caused by otitis or necrosis of the cranial bones. He described the case of a boy, aged 7, with undoubted hereditary syphilis, who suffered from squint, facial paralysis, and optic neuritis. These symptoms were followed by the development of a fluctuating swelling of the left eyelid, which was incised, and necrosis of the floor of the orbit found to exist. Complete recovery ultimately occurred. Gummata were not so rare in children as generally supposed, and he had a case of gumma of the tongue, in a child aged 3, under his care.

Dr. HADDEN, in reply, said that he had no intention of discussing all the possible aspects of this question. He believed that intra-cranial gummata in children were rare. Dr. Barlow's remarks bore out the suggestion that the case of double hemiplegia with two distinct attacks and the case of hemiplegia supervening in idiocy were of syphilitic origin. Pathological facts were wanted in order to clear up many of the points raised, and the discussion would not have been in vain if clinical and, more particularly pathological, inquiry into this subject should be stimulated.

---

*November 28th, 1892.*

## ATHLETIC EXERCISES AS A CAUSE OF DISEASE OF THE HEART AND AORTA.

By WILLIAM COLLIER, M.D., F.R.C.P.

I WISH this evening to direct attention to the growing tendency on the part of young Englishmen to indulge in certain athletic exercises to an extent which, I believe, must prove harmful. Fortunately for my purpose, medical writers in recent years have more fully recognised the part played by muscular effort in the production of heart disease than was formerly the case, and I am thereby enabled to bring forward strong collateral evidence in

support of my suggestion. One of the first English writers to recognise the connection between oft-repeated muscular effort and diseases of the heart was Dr. Peacock, who, in an interesting paper on the diseases of the Cornish miners, pointed out that these men were peculiarly liable to diseases of the heart and aorta, which he attributed to the great muscular strain necessitated by their work in wielding heavy hammers throughout the day, coupled with the fact that at the end of it they were exposed to an even greater strain, in being compelled to spend an additional hour in climbing almost vertical ladders, which was the only means they had of leaving the mines. He observed that their symptoms usually began to develop at about the age of 40, the most pronounced being slight dyspnoea and palpitation, which steadily increased in severity, so that at the end of another five years or more the majority of them were compelled to give up working in the mines; the pathological changes most frequently found were dilatation and hypertrophy of the ventricles, with, in a certain percentage of cases, incompetence of the aortic valves.

In 1870 Dr. Clifford Allbutt published in the St. George's Hospital Reports for that year a paper on the effects of overwork and strain on the heart and great blood-vessels; his experience was gained by observing the effects of long-continued muscular effort on the labourers in the forges, docks, and engineering works of Leeds. He showed that the constant strain thrown on the hearts of these men first produced dilatation of the chambers, followed by hypertrophy of their muscular walls. In badly fed and badly developed men dilatation was the most pronounced change, and often led to valvular incompetence early in the day; more frequently, however, dilatation was accompanied by hypertrophy, and for a time all went well; it was, he insisted, the aorta that suffered in these cases, the blood from the hypertrophied heart was constantly thrown into the aorta with abnormal force, and this vessel, having no power to strengthen itself, gradually lost a certain amount of its elasticity, and became stretched and dilated; this change increased the capacity of the vessel and permitted a larger quantity of blood being thrown into it at each contraction of the ventricle; in order to force it onwards the heart was compelled to beat with increased power. So, little by little, the distension was increased, until the day arrived when incom-



petence was set up, either by stretching of the orifice from dilatation of the vessel, or from the inability of the valves to support the column of blood above them. In addition to the above, atheromatous changes were often set up and aided in establishing valvular incompetence. It is important to note that Dr. Allbutt expressly states that these changes were, as a rule, very gradual, as the symptoms they induced were not sufficiently marked to make the sufferers seek medical aid until they had attained the age of 40 or upwards. In addition to the authorities I have mentioned, many writers in recent years connected with the Army Medical Department have dealt with strain as a cause of heart disease, but as they attribute the strain not so much to muscular work as to the tight clothing soldiers are compelled to wear, or to the nature of their drill, I shall not allude to their papers. I find, however, that Dr. Da Costa, of Pennsylvania, in 1871, published an interesting paper in the 'American Journal of Medical Sciences,' on "Irritable Heart," in which he asserts that dilatation and hypertrophy, accompanied by dyspnœa, palpitation, and pain over the cardiac region were frequently induced among the American soldiers by repeated long marches, and that in many instances the symptoms persisted in spite of all treatment, and rendered the men permanently unfit for service. Apparently the same kind of changes are brought about in the lower animals by precisely the same causes, for, in reply to enquiries I have made, Mr. George Fleming, the eminent authority on all veterinary subjects, tells me that hypertrophy of the heart and diseases of the aorta occur with especial frequency in racehorses, hunters, greyhounds, and foxhounds; all animals in which great muscular efforts are frequently repeated. You will some of you remember that at death the heart of the celebrated greyhound "Master McGrath" was found to be enormously hypertrophied, and Mr. Fleming records the fact that the heart of the celebrated racehorse "Helenus" weighed  $15\frac{1}{2}$  lbs., instead of about 9 lbs., the normal weight. In short, ample evidence exists which points to the conclusion that frequently repeated muscular effort is a potent agent in the production of heart disease. I shall now endeavour to show that bicycling and long-distance running are by many in the present day indulged in to an extent which is extremely likely to bring about the changes in the heart and blood-vessels referred to above. Twenty years ago the late Dr. Morgan, of Manchester, made an exhaustive



enquiry into the after-health of the competitors of the Oxford and Cambridge boat-races from the year 1829 to 1869; so thorough was his enquiry that he was able to obtain the personal experience of 251 out of 255 of the oarsmen who were still living, and, after carefully weighing all the evidence brought before him, the conclusion he formed was, that the vast majority of these oarsmen were benefited rather than injured by their exertions, that as regards heart disease there was little appreciable difference in the mortality from this cause among University oars and that which prevailed among other classes of men at a corresponding period of life. It is well to remember that Dr. Morgan was dealing with a picked number of men, exceptionally well developed, the majority of whom had undergone a medical examination to determine their fitness for the struggle, and who only competed after a prolonged and careful training. There is one other point which is of even more importance; it is that the number of contests these men engaged in during the year was comparatively small. In addition to the inter-University race, they would probably row in their college races during the May term, and some of them at Henley a month or two later. Dr. Clifford Allbutt, to whose paper I have already referred, extended his enquiries to the Universities, and tells us that Drs. Acland and Rolleston at Oxford, and Drs. Paget and Humphry at Cambridge, were strongly of opinion that diseases of the heart, as consequences of rowing and other sports, were very rare. Professor Humphry believed that this immunity was in a large measure due to the good food the undergraduates enjoyed, compared with the food of those classes to which reference has been made. I think it will be generally admitted that physicians resident in the Universities would not be, and are not, likely to see much of the evils produced by over-exertion, inasmuch as the changes are slowly induced and symptoms not likely to become troublesome till years after the injury has been inflicted and the men have gone down. Experience teaches us that sudden and violent injuries to the heart, such as rupture of a valve, the establishment of a leakage of the valves, during athletic competitions are extremely rare. Both Dr. Peacock and Dr. Allbutt expressly state that their patients did not usually seek medical advice until they were 40 or upwards; the opinions, too, of the physicians whose names I have mentioned were chiefly based on the effects of rowing, as long-distance running and bicycle racing

had not become popular forms of exercise at the time they wrote. I believe, too, that rowing men expose themselves to less risk than do the long-distance runners and bicyclists, chiefly because rowing men are not tempted to repeat their racing efforts with anything like the same frequency as are the runners and bicyclists.

It is in the frequent repetition of severe muscular effort that the danger lies. Let me remind you that it is within comparatively recent years that annual athletic gatherings have been established in most of the large provincial towns and many of the small ones. In order to make these gatherings attractive, the custom has grown up of offering prizes of considerable value to all comers, and, to induce each competitor to do his best, the open races are all carefully handicapped, the best runners having to give their weaker brethren often very long starts. The value of the prizes has been sufficient to create a class of athletes who make running almost a profession, and who travel all over the country, competing in these long-distance races on an average once or twice a-week all through the season, perhaps six months in the year. But of all forms of athletic exercises, the greatest danger lies in bicycling. This form of athletics is still in its infancy, some of the first recorded races taking place at the Crystal Palace in 1869. Since then they have become extremely popular with the public, and, as a consequence, the number of race meetings proportionately frequent. One inducement to competitors has been the very valuable prizes offered and the long starts given to inferior men; another, the unhealthy notoriety attending these competitions.

The successful cyclist finds his smallest achievements chronicled in not one, but many, of the sporting papers, and, as his friends echo the applause of the press, in time he learns to regard himself as a public hero, and to consider his success on the path of more importance than his business or profession. The result is that we find a large number of men riding on an average one or two hard races a-week all through the season. From a careful examination I have made of some of the records of prominent riders, I have no hesitation in saying they will be found to ride in from thirty to forty races at least during the year.

In addition to path racing, an unfortunate craze has risen of riding long distances against time, largely fostered by the notoriety given to it by the press. Every large club has its



record for the 100 miles, and this distance is a favourite one with them for their annual road race. Some time since I witnessed the start for one of these races, in which from fifteen to twenty candidates took part, many of whom were small chested, badly developed, badly nourished men, who must have derived injury rather than benefit from so severe a race. Every aspirant to honours in long-distance riding in the present day must try one or other of the long-distance rides against time, such as from London to York, 196 miles, London to Edinburgh, 397, London to Brighton and back, or what is known as the end-to-end ride, that is from Land's End to John-o'-Groats, a distance of 861 miles. It was considered a remarkable performance when, in 1874, the late Hon. Keith Falconer covered the distance in 12 days, but since then innumerable attempts have been made to shorten the time, and with such success that in recent times the whole distance has been covered in something under 4 days. One of the most persistent riders of this journey was Mr. G. P. Mills, who, in 1886, completed his ride in 5 days and 1 hour, spending only 6 hours in sleep; a month later he rode a tricycle over the course in 5 days 10 hours. In 1891 he attempted to lower his own record, and to do without sleep altogether; in spite of bad weather his efforts were successful, as he covered the distance in 4 days 11 hours, and had it not been for the fact that when but half-a-dozen miles from home he was accidentally thrown from his machine, and on reaching the ground fell at once into such a deep sleep that his friends were unable to rouse him, he would have reduced this time by several hours. This year the journey has been completed in 3 days 23 hours. In addition to these rides, a spirited rivalry has for many years past been carried on among prominent riders, stimulated to a large extent by the bicycle manufacturers for advertising purposes, as to the greatest number of miles that can be ridden in the 12 or 24 hours. In 1890 three members of one club, in one of these contests, succeeded in covering upwards of 300 miles in the 24 hours, and this season the distance has been increased to 413 odd. If now we accept the teaching of Dr. Clifford Allbutt, Dr. Peacock, and others, we must conclude that the excessive indulgence in running and bicycling, which I have shown to exist, is a serious source of danger; for although the muscular strain is not as continuous as was in the cases referred to by either Dr. Peacock or Dr. Allbutt, it is for the time



being much greater. It is true that I am unable to bring forward a number of striking cases to support my contention, for even if the athletic exercises indulged in at the Universities are a cause of injury, we should not expect the symptoms to develop until years after the men had gone down, but I am inclined to think that as a body University men suffer much less than others, and for these reasons, the majority of them are men who throughout life have enjoyed exceptional advantages: well nourished and well cared for from their earliest childhood, they have spent some hours daily in muscular exercise of one sort or another. But this is not the case with all athletes; in the large towns numbers of them enter business at an early age, and spend the greater portion of the day in the close atmosphere of the office, their time for training is limited to the early mornings and evenings, under such conditions they are far more likely to suffer than their more fortunate brethren. We must remember, too, that, with few exceptions, University athletes take long periods of rest between their contests, four or five weeks of training will be followed by five or six of rest; the men to whom I have specially referred are in training six months of the year, and racing once or twice a-week during this period.

Although I am unable, as I confess, to cite cases of marked injury to the heart and aorta as the result of athletics, I have had ample opportunity at Oxford during the past few years of observing the early changes in the heart induced by them. I am constantly consulted by undergraduates in all stages of their University career as to their fitness for rowing, running, or football, or because they have experienced symptoms which have made them uneasy about their hearts. In a few instances the symptoms complained of have pointed to rapid dilatation of the right ventricle, the patients seeking advice on account of attacks of giddiness, palpitation, and dyspnoea on exertion, all the physical signs pointing to a dilated right ventricle, but almost invariably such patients were badly developed, narrow-chested, weakly men. The most characteristic and most uniform, and the most unmistakable change I have found is that of hypertrophy of the left ventricle. The undergraduate who has gone in enthusiastically for athletics at school, and has continued them at the University, will have his apex beat almost to a certainty markedly displaced downwards and to the left, there will be distinct accentuation of the second sound

over the aortic area, and the pulse will be characteristically slow and strong, pulse rate nearer 60 than 70. Such a man will at times consult me, because after a severe race he has been troubled with a constant dull pain over the cardiac region, and a feeling as though his heart was being compressed, and that there was not room enough for it in the thorax. No doubt this hypertrophy is accompanied by a certain amount of dilatation of the ventricle. Side by side with it we find distinct evidence of emphysema of the lungs. In fact, I believe that hypertrophy and emphysema, by enabling the blood to become re-oxygenated with greater rapidity, is for the time being a great advantage to the athlete, but we can easily understand how such a powerful heart, if stimulated too frequently by great muscular efforts will gradually produce stretching and dilatation of the aorta, so clearly described by Dr. Clifford Allbutt. Athletes, with their dilated hypertrophied hearts, run another risk, I think. Many of them, after several years of hard athletic work, suddenly give it up and lead sedentary lives, only indulging in hard exercise at long intervals during their summer vacation, and it is on these occasions that they incur the risk of bringing about rapid dilatation of the ventricles, for during their long periods of rest, while the cavities remain dilated, the hypertrophied walls undergo a certain amount of atrophy, grow weaker, consequently when the extra strain suddenly falls on them, they are unable to cope with it. I believe this will explain the fact that often a man who was a great athlete in his youth, in middle life is not able to hold his own in athletic exercises (such as walking, and especially mountaineering) with friends he could easily surpass in his younger days.

Living, as I have done, for the greater part of my life in one or other of the University towns, no one could be more impressed than I am with the value of athletics, if carried on within the bounds of prudence. As I believe these bounds have been largely exceeded in recent years, I am particularly anxious this evening to elicit the opinion of others who have better opportunity than I have of confirming or disproving the fears I have expressed, and especially I am anxious to learn whether the marked enlargement of the hearts of athletes to which I have referred is to be regarded as a morbid condition or not.

Dr. SANSOM remarked that no symptoms of circulatory disturbance could be observed in many athletes, who, nevertheless, showed evidence



of hypertrophy of the left ventricle. In coming to a conclusion whether this hypertrophy was to be regarded as physiological or pathological, the muscular development of the subject must be taken into consideration ; when a large muscular system was associated with a large heart he was disposed to regard the condition as normal. He had, however, examined athletes who showed signs of dilatation of the right heart, while in others there was evidence of disturbance of cardiac innervation. He had seen a number of cases of tachycardia which were to be directly attributed to over-strain from racing on cycles against time. The condition might last for years, and structural disease of the heart might supervene. Under certain circumstances the delicate cardiac endothelium might develop a distinct endocarditis as the result of over-strain.

Staff-Surgeon PRESTON, R.N., remarked that his experience was corroborative of the statements made by Dr. Collier, and he showed how that, by the introduction of mastless vessels into the Royal Navy, and by the substitution of hydraulic and steam powers for the hand-working of heavy guns, cardiac diseases had considerably diminished amongst the men serving afloat. There was, he continued, no form of athletic exercise so severe as sail-drill, and at no period of a seaman's life was his circulatory system more severely taxed than when drilling aloft in time-competition with other vessels. That such emulation was physically injurious had been admitted by the executive, and was now forbidden, with the results that the ratio per 1,000 of admissions to the sick list for diseases of the circulatory system had been reduced from 7.94 during the ten years 1875-1884 to 5.8 during the period 1885-1891 ; the latter period marking the introduction of mastless vessels and hydraulic gun-power. The reductions in the invaliding and death ratios had been equally satisfactory. Systematic gymnastic exercises, under medical supervision, had recently been introduced in H.M. Navy with admirable results, but no undue strain upon the heart or great vessels was permitted ; while boys, under training as oarsmen, were carefully watched, and under no circumstances allowed to overtax their growing frames. The conditions under which the land forces of the Royal Marines undergo physical instruction were also commented upon and compared with those existing in the Royal Navy. It was shown that no change has taken place in the ratios of men under treatment for diseases of the circulatory system for many years, the strain upon the system remaining unaltered as far as the official routine of drills, gymnasium, and marchings was concerned. Staff-Surgeon Preston also remarked that, in his opinion, men who were the subjects of acquired constitutional syphilis were wholly unfitted to stand the strain of athletic exercises, the specific poison having already weakened the reserve power of the heart and predisposed the aorta to dilatation.

Brigade-Surgeon HAMILTON said that aneurysm had become rarer among soldiers of late years, but since the introduction of quick manœuvres the "irritable heart" had become far more common ; it had also been produced in the cavalry by riding without stirrups. He regarded a man with secondary syphilis as practically incapacitated from service. Functional disorganisation of the heart was also materially contributed to by the abuse of tobacco and alcohol.

The PRESIDENT did not take such a gloomy view as to the results of syphilis. For treatment he much preferred small doses of mercury, which was much superior to prolonged administration of iodide of potassium, which undoubtedly had a most depressing effect on the muscular system.

Dr. COLLIER, in reply, said, with reference to the influence of syphilis



he was not in a position to speak, as the undergraduates of Oxford did not often expose themselves to the risk of contracting that disease. He wished to emphasise what he had said with reference to the changes which took place in the heart when athletic exercises were abandoned, which rendered that organ unable to cope with sudden strain.

## PILES: THE IMPORTANCE OF RECOGNISING THE VARIETIES AS DETERMINING THE SELECTION OF TREATMENT.

By HERBERT ALLINGHAM, F.R.C.S.

IT is with no slight trepidation that I venture to bring such a simple subject before this Society to-night. I am, however, induced to do so from observing that there appears to be in the minds of some a great confusion about this malady, there being no distinct appreciation of the varieties of piles and the varieties of treatment which are required under different conditions. For instance, it is absolutely absurd to think that all piles can be cured by physic, and it is equally absurd to hold that all piles require the knife to effect a cure. In order that medicine and surgery should play their proper part in these affections, it is most desirable to bear in mind the varieties of piles and the different constitutions of the patients. I do not intend here to enter into any minute anatomy of piles, but to discuss them purely according to their clinical conditions.

Careful examination is always most necessary, for it is constantly said a patient has hæmorrhoids when, as a matter of fact, he is suffering from pruritus ani, fissure or ulcer, polypus, or even a blind internal fistula, &c. All these I have seen mistaken for piles, and, obviously, to treat these complaints as piles is utterly useless.

Piles may be classified as follows:—

External.

Internal.

Externo-internal.

A thorough appreciation of these conditions is needed before deciding whether medicine or ointments will be sufficient to cure or whether surgery is to be called into use.

*External piles*, strictly speaking, are of two kinds—

1st. A small piece of loose skin which may become inflamed and even suppurate.

2nd. A small varicose vein which, in like manner, may become inflamed or suppurate.

An *attack* of external piles arises from inflammation of the tabs of skin or from the varicose vein becoming thrombosed. This state of things may be started by constipation, not sufficient exercise, or even too much exercise, excessive drinking or eating, and many other causes. All that is requisite when this is the case is to correct the irregularity, give medicine to relieve any congestion of the liver, and apply to the anus some soothing ointment or lotion. Should the pain be great and the little tabs or thromboid veins commence to suppurate, they may be freely incised or even cut away. This, however, is seldom necessary, for these inflamed external piles rarely suppurate, and with laxatives, local sedatives, and rest are cured in a few days.

*Internal piles*, for practical purposes, should be divided into venous, arterial, and capillary. This division will probably be disputed, for many people, from reasons based on minute anatomical researches, deny the existence of these varieties. Still, for practical purposes, and after all that is the most important point, these conditions exist and must be taken into account.

*Venous piles* are, as a rule, large bluish tumours which prolapse at stool, and in advanced cases, when the sphincter becomes relaxed, may come down any time ; they bleed occasionally, sometimes very freely. This variety is, no doubt, caused by some stagnation of the venous systems brought about by constipation, by excessive drinking or eating, by certain cardiac or pulmonary diseases, or by any intra-pelvic pressure. This class, in the early stages may be greatly relieved, in fact, frequently cured, by judicious medical treatment, viz., by laxatives, regulations in diet and drink. But it must be borne in mind that if these piles have existed long and the veins have been strained, and so permanently dilated beyond recovery, an operation is the only mode of effecting a cure. In this stage drugs are useless, if not even harmful.

*Arterial piles* are more generally found in the young adult, and are not, so far as can be made out, due to any portal congestion or other irregularities. They may be, and no doubt often are,

hereditary. They are in the majority of instances smaller than the venous variety, and are reddish in hue. They do not, as a rule, prolapse so much as the venous kind, but they often bleed at stool, at times very profusely. From the number of cases I have had the opportunity of watching at St. Mark's Hospital, I am of opinion that in the treatment of this variety little or no good is to be gained from physic, an operation being the only method of permanently ridding the sufferer of the trouble.

*The capillary pile*, if it may be so-called a pile, is a rarer condition. In this class of cases, there are not any definite tumours, but only vascular areas, which are very slightly, if at all, raised above the mucous membrane. These are almost nævoid in character. There is no assignable cause for this variety. There is very free bleeding at times, but little other trouble. Cessation of bleeding may be obtained by astringent ointments, but should this treatment fail after a fair trial an operation is then necessary.

*An externo-internal pile*, as the name denotes, is an internal pile merging into an external one. On examining a patient so affected nothing is to be seen except the external pile, but on gently pulling upon this an internal pile will slip down and become exposed, the external one running into and becoming directly continuous with the internal one. This state of things may be aggravated by constipation, &c., and any physic that relieves this will improve the condition of the pile. However, no absolute cure will be made without an operation.

All these varieties of internal piles, with the exception of the capillary, may become prolapsed and inflamed, and even suppurate. When this is the case a mild laxative should be given, and some warmth or sedative ointment should be applied until the inflammation has subsided. This protrusion and inflammation, with possible suppuration, is Nature's attempt at cure, but being, as a rule, a very feeble and imperfect one, an operation later on is required to complete the cure.

External piles are generally cured by medicine, and proper unguents or lotions. Various internal piles, if the patients have not had them too long, may be cured by medicine, &c. Capillary piles may be cured by astringents applied. In bad or old-standing cases of venous piles, external piles, capillary piles not giving way to astringents, and the externo-internal piles, operation is necessary.



Having decided an operation is requisite, a choice must be made between the various methods which may be employed.

They need not all be enumerated, as many of them are obsolete.

Those worthy of mention are—

Ligature with incision.

Crushing.

Clamp and cautery.

Excision.

*Ligature with Incision.*—Briefly, this is as follows:—The pile is drawn down by a vulsellum, and is separated with scissors from the muscular and submucous tissues upon which it rests. The incision is made at the junction of the skin with the mucous membrane, and is carried up the bowel, so that the pile is left connected by vessels and mucous membrane only. A strong silk ligature is then tied as tightly as possible at the neck of the pile, and great care is required in tying all the knots that are made, as many as three being desirable. The ligatured pile is then returned within the sphincters. The pile is not to be transfixed by the ligature, that is quite unnecessary.

*Crushing* consists in drawing the pile by means of a hook with a powerful screw crusher, which is tightly screwed up, and the distal portion of the pile then cut off. The crusher should be applied on the longitudinal surface of the bowel, and should be left on about two minutes. The power exerted is very great, and completely crushes the vessels in the stump, thus preventing bleeding after its removal.

*Clamp and Cautery.*—The pile is drawn into a clamp, the distal portion is cut off, and the stump within the clamp is cauterized until all the vessels are thoroughly seared.

*Excision.*—The pile is seized, and is then cut off at its base with scissors, any bleeding vessels being secured.

I must state without reserve that *ligature with incision* is the best of all operations: it is the one that has been practised at St. Mark's Hospital for many years, and has always proved superior to other methods. It is the quickest to perform, little blood is lost at the operation. There is little or no fear of secondary hæmorrhage. It is certainly the safest in all cases of very bad piles, or when the patient is excessively anæmic, or when it has been necessary to operate in those affected with visceral or arterial

disease, and, again, it is the safest when the patients have to be left some distance from medical aid.

*Crushing*, in my opinion, is the next best method, in that, as a rule, there is less pain following the operation, and the patients get well more speedily than after the ligature, there being no sloughs to come away. This operation should be performed in healthy subjects when the piles are not very large or vascular, and are rather pendunculated. It is more applicable in women where the bowel can be commanded by the finger in the vagina, should there be any vessels bleeding at the operation.

*Clamp and Caутery*.—The two former operations presenting such advantages, there is nothing to be gained from the use of the clamp and cautery, which is a barbarous proceeding. There are many points against it. It is certainly more painful than the above methods. It is at least six times more fatal. There is more likelihood of secondary hæmorrhage when the burnt sloughs separate. There is a greater tendency to contraction after the operation, since a burnt scar is more liable to contract than any other one.

*Excision of Piles* is one of the oldest operations, and is useful in simple cases, or where there is but one pile. This leads me to Mr. Whitehead's operation, which is merely an elaboration of the simple excision method. Mr. Whitehead holds the theory that there may be a pile area in the rectum, and infers that because a patient has three or four piles in the rectum, all the lower end of the gut is diseased and should be removed. It might as well be argued that because a patient has a nævus in his mouth all the mucous membrane of that cavity should be excised. I have seen many patients after Whitehead's operation, and have observed that contraction is a common after-result, and so, too, is ulceration. There are other disadvantages: a large amount of blood is lost at the operation, almost as much, indeed, as when the rectum is excised for malignant disease. The operation, instead of taking at most ten minutes, genererally occupies about three-quarters of an hour to complete.

If piles are properly removed by ligature, &c., there is no likelihood of their ever returning. We sometimes hear of cases in which piles are said to recur: that simply means that in the first instance they have not been thoroughly removed.

There are certain points to be considered which depend less

upon the variety of piles than on the patient's age and position in life.

We might take the case of an old and wealthy man, to whom loss of time is of no importance. Say, in addition to his piles, he has some visceral complaint. It might be wise for him not to run even the smallest risk of any operation, however slight it might be, but rather to resign himself to a course of perpetual treatment by drugs, to rest after each action of the bowels, and to daily use injections. Even in a case like this, abstention from operative treatment would be altogether wrong if the piles bled badly.

Far stronger are the reasons for operative measures in another typical instance. The patient is young, and his piles prolapse. Taking drugs and local treatment may benefit him for a time, but do not cure him. What will be the result if he continues to take medicine, lies down for an hour or so, and has to administer an injection to himself after every action of the bowels, to say nothing to having to apply ointments and lotions daily. He will soon become a confirmed invalid. The strongest possible protest must be made against such a course of treatment and its consequences. The only sound and honest course to pursue is to advise the removal of the piles once and for all, if the operation is thoroughly done. The patient will then be restored to a natural state of life, and be freed from the dangers of becoming valedudinarian.

To return to the general principle of this paper, the high importance of distinctly appreciating the particular kind of pile from which the patient is suffering.

In some varieties which I have mentioned medical treatment is sufficient to cure, and should certainly be employed. For the other varieties it is absolutely futile to a continuance of such treatment in lieu of a resort to operation, and is unjust to the patient from every point of view.

The PRESIDENT said that his experience confirmed his preference for Salmon's operation. He always thoroughly dilated the sphincter and so as to obviate pain after operation.

MR. SWINFORD EDWARDS said that he could not agree with the author's remarks concerning the treatment of external piles. For his part he considered that some small operation was generally to be recommended, such as, in cases of thrombic piles, a simple incision for the purpose of turning out the clot, and, for loose tags of skin, simple removal with scissors. It was most important for the patient to be rid of these external piles, as they were often the starting point of a small fistula, and pruritus



ani not infrequently resulted from them, or was, at least, kept up by them. With regard to the three classes of internal piles, although he (Mr. Edwards) recognised the varieties described by Mr. Allingham, he considered that the particular treatment to be adopted should rather depend upon the symptoms to which they gave rise, than upon their anatomical appearance. As a preliminary to all operative interference, he strongly advocated a thorough forcible dilatation of the anus. The usual operation practised by himself was that of ligature, or what is generally known as Salmon's operation. In this operation great care should be taken, if the ligated piles are cut off (a proceeding abandoned by himself) to leave a sufficient long stump, or the ligature may slip and give rise to very serious recurrent hæmorrhage. All small bleeding vessels in the outer side of the incision should be secured, or they may occasion considerable bleeding into the rectum after the parts have been returned. The method of crushing is perhaps the best where time is of great importance: two of his cases having left England for abroad eight days after the crushing apparently well, moreover no wound could then be seen or felt in the bowel. In conclusion, he said that the treatment by injection of carbolic acid was not known perhaps so widely as it might be. He had a pretty large experience of it, having thus treated 120 cases. Its advantages are that no confinement to bed and no anæsthetic is necessary. The patient commences to improve immediately after the first injection, and is able to attend to his business during the whole period of treatment.

Mr. BIDWELL spoke in favour of Whitehead's operation. He considered it a more surgical proceeding than the other methods. He saw no reason why, with antiseptic precautions, the same principles of treatment should not be applied to the rectum as to other parts of the body, where varicose veins, &c., are excised, and the resulting wound stitched up. He was sure that there was much less pain after a Whitehead's operation than after any of the other methods; in fact, a gentleman who had been operated upon, five years previously, by the clamp and cautery, was much impressed by the comparative painlessness after a Whitehead's operation, which had to be performed this year, on account of a recurrence of his piles. The loss of blood is not considerable, and the operation should not last longer than thirty minutes.

Dr. SOLOMON SMITH asked whether it would not be possible to extend the domain of medical treatment, and, by the adoption of the simple procedure of dilating the anus, bring within its boundaries many cases which otherwise would have to submit to the more severe operative measures. In a disease like hæmorrhoids, some cases of which were so severe as clearly to necessitate operation, while others were perfectly amenable to simple medicinal and dietetic treatment, it was obvious that a borderland must exist, and that cases must occur in which the question of operation might be somewhat doubtful. It was worth consideration then whether some of these cases might not be brought well within the reach of simple treatment by that same stretching of the anus which seemed to be used as a preliminary to all forms of operation. By this process rest was secured to the part, and that spasmodic contraction of the muscle was prevented which was not only provocative of much of the pain of piles, but was one of the main causes of their congestion by preventing the return of blood from them. In many cases piles were perfectly innocuous for long periods of time, and, although many severe cases required radical operation, it would probably be found that in a considerable number of

instances, even when painful and bleeding, they could be brought back to that harmless condition, in which a very little simple treatment kept their owners in perfect comfort, by the process of stretching the sphincter, a proceeding which, he believed, was being used by some surgeons for that very purpose.

---

*December 5th, 1892.*

## CHOLERA, ITS EPIDEMIC PROGRESSION AND CAUSATION.

By Brigade-Surgeon-Colonel J. B. HAMILTON, M.D.

THE first great difficulty that strikes the investigator when he comes to consider the subject of cholera, is the number of theories, each conflicting with the other, and each put forward confidently and with dogmatic assertion, and, no doubt from the standpoint of the various writers, in perfect good faith as to their correctness.

Each local observer necessarily receives different impressions in accordance with the facts that come under his observation, and thus we have several theories, each of them conflicting with the other, and each strenuously upheld by its advocates.

Again, we have the statistician surveying the facts connected with the various outbreaks from a central office, comparing manifold observations as regards numbers, dates of attack, and progression, and from these data working out the relation of one epidemic to another, till he has indications sufficient to map out the laws that appear to indicate the progressive steps of the disease.

This is especially the case in India, and, no doubt, had anything like unanimity prevailed among the recorders of cholera as it appears in that country, much greater weight would have been given to their writings and reports.

Unfortunately, exactly the opposite is the case, and it is impossible to consult the numerous authors of books on cholera without at once becoming aware of the great diversity of views held by different writers.

This fact has, I am convinced, gone a long way to discredit Indian opinion on the subject, and we, who have spent many years

in the midst of the disease are not, as we ought to be, regarded as reliable authorities on the subject.

In fact, there is rather an impatience shown by the profession at home against Indian medical officers when they attempt to put forward their views, chiefly, I believe, for the above reasons.

My object in reading this paper is to attempt to show that no theory hitherto propounded will cover all the ground of epidemic cholera, and that in every case the explanation given falls short in some material particular.

The theories usually put forward to explain outbreaks of cholera are mainly three, viz. :—

1. Propagation by drinking water, food, &c., into which the poison has entered.

2. Human intercourse.

3. Conveyance of the disease by the air.

We have now added the so-called vibrio, or cholera bacillus, which has latterly taken possession of most European authorities, and against which it is almost a heresy to say a word.

Before I proceed further, there is one point worthy of consideration, viz., whether cholera as seen in Asia is identical in every respect with cholera as observed in Europe? I think I am correct in saying that all writers and observers agree in considering cholera identical from a clinical and pathological point of view, both in India and Europe, so that the only questions to be settled are, is the epidemic progression the same, that is, are the influences that spread the disease similar in both countries, and are the actual causes of individual infection identical?

No doubt the climate of Europe is vastly different from that of India. Still we see the disease in both countries prevalent at the same season, viz., in summer, that it dies out as winter advances, and that warm damp weather is generally favourable to its development.

We also observe outbreaks of cholera occurring along certain well-defined lines, and becoming localised in certain towns and tracts of country; also that the disease is most intense and fatal in the commencement of an epidemic, gradually becoming less virulent till most of the later cases recover. (This fact accounts for the number of the so-called specifics which are vaunted by various persons, but which when tried in the commencement of the next epidemic fail signally.) We also see that as a rule cholera does



not retrograde or work backwards, though here and there recrudescences or revivifications occur, exactly as they do in the East; in fact, the behaviour of cholera as an advancing epidemic seems to be the same both in Asia and Europe. Having said so much, let me now briefly glance at the so-called theories put forward by various authorities.

First, the water theory, with which, of course, is associated the introduction of poison in food, &c.

This view seems to be the one most generally adopted, no doubt in consequence of the discovery of the vibrio or bacillus, which seems to be very generally accepted (I submit on insufficient proof) as the exciting cause of the disease.

A well-known writer in a late number of the 'Nineteenth Century Review,' in a paper on cholera, commences by saying, "Cholera is a filth disease carried by dirty people to dirty places," and then goes on to show that polluted water is the chief, indeed almost the only, cause of cholera epidemics.

In support of this, he adduces the outbreak in East London in 1866, caused, he asserts, through the accident of a broken pump and filter bed out of order, in consequence of which the water of the Lee, polluted by a cholera-stricken family, was distributed direct, unfiltered, through the mains.

Admitting for the sake of argument that the water of the Lee was so polluted, and was the actual cause of the outbreak, I would ask if it can be truly contended that the want of filtration had any appreciable effect?

For my own part, I am a total disbeliever in filters, at all events as generally used, and in India we have frequently found water that has been passed through filter beds and then through the Government filters to be far more impure than that taken direct from the well.

Anyhow, I beg to doubt the efficacy of the rough filtration in use by water companies in removing a bacillus so minute as that of the cholera vibrio.

No bacteriologist would expect to sterilise a fluid by means of any ordinary filter of commerce, much less by means of the common filter beds used by water companies.

Before I leave this part of the subject, I would like to draw attention to a most valuable paper on the "Purification of Drinking Water by Alum," by the State Geologist of New Jersey, pub-

lished in 1884. I feel convinced that such a system, if brought into general use in this country, viz., precipitating the organic matter by adding  $2\frac{1}{2}$  grains of alum to a gallon of water, and then passing it through filtering paper, or cotton, would be infinitely more reliable than the use of ordinary filters, besides being far cheaper.

To revert to the paper in the 'Nineteenth Century,' I would ask if it is not rather illogical to first assert that cholera is a filth disease, spread by dirty people, and then to show that the cleanest people, living under the best sanitary conditions in other respects, may be attacked wholesale if their water supply is polluted by cholera dejecta.

I am not, I regret to say, a bacteriologist, but I have, within the past few days, consulted one of the most eminent authorities in London, Dr. Crookshank, of King's College, and he assures me that it has never yet been proved that bacilli have caused or propagated any disease except anthrax, tuberculosis, actinomycosis, and certain septic diseases.

He further informed me that zymotic diseases (except, perhaps, enteric fever, and that is doubtful) have not been traced to a bacillus, or that these diseases can be propagated by means of bacilli.

Koch declared that he had proved the comma bacillus to be the cause of cholera in India, but Peffenkofer, Klein, and Surgeon-Major D. D. Cunningham have asserted, after full consideration of the facts and examination of the water on which Koch based his theory, that cholera was not caused by the comma bacillus. They showed that in an outbreak in Calcutta the inhabitants of certain houses who drank only the pure and wholesome water supplied by the water works suffered severely, while the villagers living close by, on the banks of a filthy tank, the water of which they drank, escaped completely, though the water in question teemed with the cholera bacilli of Koch.

Other similar instances were shown of native communities freely using the tank water in which Koch discovered the dreaded bacillus who escaped without a single case of cholera during the whole year.

It is stated by numerous sanitary authorities that there has been a marked and notable decrease of cholera in Calcutta and other places after the introduction of a pure water supply. Surely this is only

what might have been expected, and is it not the same everywhere? Introduce pure water and the public health improves. Impure water not only induces general ill-health in a population, and increases its receptivity of diseases, but it also induces diarrhœa, dysentery, and other bowel affections, the very conditions most favourable to an attack of cholera. I trust I may not here be taken as decrying in any way the great value of a pure and wholesome water supply; quite the contrary, I look on it as the greatest factor in the health of any community, but at the same time I think we should not accord to water an undue influence as a cause of cholera, lest we should be tempted to overlook other equally vital factors.

The case of Fort William, Calcutta, is also frequently quoted as an instance of the value of a pure water supply, and Mr. Macnamara in his work alludes to the improved condition of the health of the garrison as being due to that cause.

Mr. Macnamara shows that, while from 1826 to 1864 the average annual mortality among the British troops was 20 per 1,000, after the introduction of a pure *tank* supply in 1863 the mortality fell to 1 per 1,000.

I have consulted official documents on this point and find that from 1863 to 1872 the mortality was 19 for the decade, out of an average strength of 900, or 2 per 1,000 per annum, roughly.

In 1872 or 1873, the pipe water was introduced, and in the next decade the mortality fell to 10, or 1 per 1,000, while in the last ten years the mortality was 16, or, roughly, 1·5 per 1,000.

Now, this would be a highly satisfactory improvement if it could be all attributed to the water supply, but during the period under review the general sanitation was immensely improved. New palatial barracks were completed, the dry earth system was introduced and perfected, and, most important of all, on the appearance of cholera in the Fort, it is at once vacated and the troops are placed under canvas.

Surely some, if not much, of the improvement may be attributed to these changes.

However, here is a remarkable fact: in 1871 this pipe water was used in the Great Alipore Jail for the first time, and its consumption was followed by an outbreak of cholera among the prisoners, which caused a mortality equal to that of the seven previous years collectively.



Surely we might as well lay to the door of the pipe water supply the increased mortality in the jail as the improvement in the health of the troops! We know that one would be false, may not the other be also?

The history of the epidemic progression in cholera in the Bengal Presidency of India is very uniform, and, starting from the Delta of the Ganges, which is regarded as its chief endemic area, it invariably makes its way in a N.W. direction, corresponding with one branch of the monsoon current, or in a N.E. direction towards Assam with the other branch, or in both directions simultaneously.

It is most important to note that both these directions of advance are against the currents of the Ganges and Bramaputra respectively.

Put in the shortest possible way, "cholera is ever present or endemic in Lower Bengal. Heavy rainfall swamping the low-lying endemic area extinguishes cholera there, and with the monsoon current the disease advances up the valleys of the Ganges or Bramaputra, or both."

Now this fact certainly tells against the theory of propagation by water, and would incline us to regard the air, *plus* heat and moisture, as being the chief factor in progression.

To show the difficulties that beset the investigator, I may here quote an instance given by Mr. Macnamara where cholera made its way to Bombay from the East of India towards the end of the rains in 1849, in spite of the S.W. monsoon which blew during the week with a force equivalent to a velocity of 15 miles an hour, in a direction contrary to that in which the cholera advanced. These are the pitfalls which beset the steps of the investigator, and upset all theories.

We see cholera thus advancing in Bengal in a N.W. direction (I will at present disregard the N.E. current), dying down in the winter, and again breaking out and resuming its upward course as the weather gets warm, the outbreaks culminating during the monsoon. This is what took place during the great epidemic of 1878-79. An overflow from the endemic area occurred in July, and advanced rapidly to Oudh, causing great mortality. During the winter it subsided,\* but reappeared in the summer of 1879, and then the epidemic swept over the Punjab and right into

\* A few cases indicative of its advance occurring in the Punjab.

Cabul through the Khyber, carrying off tens of thousands of natives and many Europeans.

Here it is worthy of remark that while the epidemic was raging in the North-West Province and Oudh in 1878, the railways ran daily into the Punjab carrying numbers of natives from the infected districts into those that up to that time remained free, and though numerous cases occurred in the trains, and many were attacked and died far up in the Punjab, the disease never became epidemic there during 1878, and, in fact, refused to break out in the uninvaded districts.

Next year, when cholera was raging in the Punjab, in a similar way many people suffering from the disease were carried south by the railway to the country which had been swept by the epidemic in 1878; again it refused to break out in the districts that had suffered during the previous year.

Thus we see cholera advancing against the flow of the rivers, and actually refusing to ignite in uninvaded districts, or break out again in the country over which it had passed the previous year, though the bacillus (if there be one) must have been sown broadcast over the area.

To consider next the case of the Hurdwur fair. Taking Hurdwur as a centre, it will be evident that the pilgrims flock to it from every point of the compass, and that when the fair is over they return to their homes over the same routes, but these pilgrims do not carry cholera with them in every direction.

If cholera has been epidemic at the fair, the Punjab has been invaded, and those travelling north and north-west carry the disease with them to their very villages, but those travelling south only suffer for a few days, limited, apparently, to the period of incubation. In other words, those who have been infected at the fair develop the disease no matter in what direction they travel, but whereas those proceeding south lose the disease after about a week's march, those travelling upwards continue to suffer, and apparently spread the disease among the population up to the furthest limits of the Empire.

When it is asked why cholera does not retrograde, the answer usually given is, that all who were susceptible have been attacked. This seems to me to be a singularly weak argument, and not at all borne out by the usual conduct of the disease.

I ask how is this susceptibility to be determined, and how can we show who are, and who are not, susceptible?

In small-pox we know that the unprotected are susceptible.

In enteric fever we know that the young are most liable to be attacked.

The children of consumptive parents are more likely than others to suffer from tubercle; but in cholera we see no such indications of susceptibility.

In an outbreak of cholera we see the strongest and the weakest suffer equally, the temperance man and the drunkard, the healthy woman and the delicate child, the old man and the baby in arms. We also see the disease sweeping down one side of a street, or attacking the end of a barrack room; and in India we frequently observe that the inhabitants of particular barracks, even certain beds in certain rooms, are attacked out of all proportion to others in successive epidemics.

The only persons particularly susceptible in my experience are those suffering from looseness of the bowels, though it is often a subject of remark that fear seems to have a strong predisposing influence.

I consider there is no immunity, and no particular susceptibility, and, further, that one attack gives no protection against a second.

Reverting to the common idea of infection by water, I may quote some instances that came under my immediate observation, *per contra*. In 1870 I sailed in a hired transport from Calcutta with invalids round the Cape. About eight days after we sailed there was a well-marked case of cholera among the men. The ship was fumigated, and there were no further cases. The man attacked had come down by train from Upper India, which was free from cholera, and as far as humanly seemed possible was not exposed to infection before going on board.

We had a water supply taken from the Hoogly, and we drank this water for the rest of the voyage.

Again, when in charge of a hospital ship during the Burmese War of 1866, cholera broke out on board after we had been a week at sea, and we had six cases and two deaths. Suffice it to say I landed the troops, while we fumigated the ship and threw over-board every article that could possibly have been contaminated; we



had not another case! In this instance our water was distilled from the deep sea off an uninhabited coast, and we had not a drop of shore water on board.

In another instance, in the autumn of 1890, cholera had been raging in the native city of Lucknow, which lies about 3 miles to the west and windward of the infantry barracks.

It next attacked the jail, lying between the city and the barracks, and then, skipping clean over the barracks, it broke out among the enteric fever patients in a detached block.

I telescoped the ordinary sick in the next block, and transferred the enteric patients who had not been attacked to the end of this building.

Next day the disease again appeared among the same body; the ordinary sick were again closed up, and the remainder of the enteric cases moved on.

A third time the disease appeared among this body, and, finally, the few survivors were put into tents a few yards outside the hospital wards.

The disease then ceased, but the survivors nearly died from the heat in the tents, the fear of which had prevented my putting them under canvas before.

Now what could water have had to say to this outbreak?

In the first place, these patients got no water but lime water that had been in bottles in stock for weeks, and if by any possibility and against orders they did get water, it was the same water that every one else in the hospital had, taken from one well. The European orderlies, eleven in number, and about an equal number of native servants, employed in the enteric fever block, who *did* all drink water, escaped entirely, and not a single person was attacked in the rest of the hospital, barracks, or regimental lines.

It might be said the milk or food supply was the cause, but here there seemed even less probability, as the milk was taken from cows milked under strict supervision, and it, as well as the rest of the food, was common to all in the hospital.

I may here say that the supply of potable water for the European barracks is remarkably good, and no pains are spared to keep it pure and free from pollution.

Certain wells are selected, the water is carefully analysed by experts, and, if found pure, the selected wells are covered in, only

a tube through which the water is pumped being allowed to enter them.

The water is then passed through a filter bed, and finally through Macnamara's filters. Yet, with all these precautions, it not infrequently happens that cholera attacks the European barracks, while the filthy native bazaar close by, with its water supply resembling liquid sewage, and contaminated in every possible way, escapes without a case.

I had written so far of this paper when the account of Pettenkofer's experiments on himself, and also of those of Professor Emmerlich, appeared in the 'British Medical Journal.' As will be remembered, these gentlemen, having first neutralised the gastric juice with bicarbonate of soda, each swallowed a cubic centimetre of fresh bacillus cultivation, with the result that after two days they were attacked by colic and diarrhœa, though otherwise feeling perfectly well.

I would here draw attention to the remark that both these Professors are stated to have suffered from colic. Now, it is an admitted fact that the premonitory diarrhœa of cholera is a painless diarrhœa, and in this lies its great danger.

This fact goes a long way to prove that neither had an attack of Asiatic cholera in any form, though it is quite possible that the milliards of bacilli made themselves very unpleasant before they were finally got rid of.

Similar experiments have been carried out in India with the same results, and this fact seems proved, viz., that the comma bacillus alone is not capable of producing cholera in a healthy person, even when that person is specially prepared by having the gastric juice neutralised, *unless there is some other factor present in addition.*

Whether the vibrio or comma bacillus is a factor in producing an attack of cholera seems to be still an undecided question, though the evidence appears to bear out the statement that it is always present in the dejecta.

Admitting that the bacillus is a factor, and the evidence seems to show it is, that it is not the only factor I am convinced, but what the other factor or factors may be is still hidden from us.

For the general public the theory of a bacillus may suffice, and it should be our duty to show them how to prevent the multiplication of this factor by sanitation and attention to the public health,

but for the profession of medicine to rest satisfied with our present knowledge would be contrary to all our best traditions.

We are, so to speak, on the track, and perhaps Pettenkofer's equation is closer to the truth than has been generally admitted. As you know, Pettenkofer says that " $x$ " represents a specific germ disseminated by human intercourse, that " $y$ " is a factor dependent on time and place, and " $z$ " is the individual predisposition.

The " $y$ " seems to be the difficulty, and whether or not it is some peculiar condition of the atmosphere, which seems possible, we have yet to discover.

In the present state of our knowledge it would be absurd to assert that polluted water, food, &c., can have no influence in causing outbreaks of cholera. At the same time I am convinced that water in which the vibrio or comma bacillus is present will not of itself alone cause cholera. There is a "something," an "entity"—what it is I know not—that must be present in addition; in short, there must be this influence, in addition to polluted water or food, to cause an attack. This may seem to be an empirical statement, but I know it is a view held by many observers in common with myself, and is indeed almost identical with Pettenkofer's equation.

I must next briefly allude to "human intercourse," which has been adopted by many writers on the subject as a creed.

First, what are we to understand by "human intercourse" causing cholera?

If it is infection from the sick to the healthy, it is certainly incorrect, for if one thing more than another has been proved regarding cholera, it is its non-infectious or contagious nature, so far as the words infection and contagion are generally understood.

Personally I can state that I have never seen cholera break out among attendants, though of course now and then it happens that they are attacked, but not out of proportion to the rest of the population.

In one great epidemic in India, of which careful records on this subject were kept, it would appear from the returns that the safest places to reside in were the wards of cholera hospitals.

On the other hand, there is much evidence of communicability from man to man under certain conditions.

It has frequently happened that troops when moved into camp



carry cholera along with them, and so well is this understood, that they are always broken up into as small bodies as possible, and the camps are moved at right angles to the wind on the occurrence of fresh cases.

Even under these circumstances we see cholera cleaving to a body of men and troops, even conveying it with them by train, while the villages near which they encamp, and from which they obtain their water supply, may remain free. In fact, it would appear as if a body of men carried about with them some infective material which keeps the disease alive notwithstanding all precautions.

A very remarkable instance of what was looked on as "human intercourse" occurred at Allahabad last year. I was P.M.O. of the district, and am able confidently to state that up to the morning of the 18th of May there was not a single suspicious case among the European troops, not even one of diarrhœa. At 7 A.M. on that date cholera broke out in the S.W.B.'s (24th Regiment), and, to make a long story short, in four days we had altogether, out of some 800 men, 53 cases and 32 deaths.

There was absolutely no insanitary reason for the outbreak: the barracks, water supply, and food all being in perfect order. I may also mention that the prisoners in the cells were the most severely attacked of all, and several died, while not a single person in, or connected with, the hospital was affected.

I at once had the regiment moved out into camp by rail 40 miles off, and placed under canvas on a bare plain on which cholera camps are always pitched, as the site has been singularly healthy. The heat was terrific, 110—115° in the tents, yet there was not a case of sunstroke.

During the first 24 hours after going into camp there were 9 cases, in the next 24 hours 5 cases, and on the third day 3 cases, but after that the outbreak ceased.

This would seem to prove that the period of incubation is limited to three or four days, as almost certainly these men were infected in barracks.

I may mention that a battery of Royal Artillery occupying the same lines was sent into camp as a precaution, and had not a single case.

Now, what was the cause of this outbreak? It was confined to the regiment, and the native troops and civil population escaped

at the time, though the regimental bazaar and servants of the corps left behind suffered severely.

We knew that cholera was epidemic across the Ganges a few miles off, and the natives from the villages in that district came in daily, carrying fruit, vegetables, grain, &c., for sale. The road lay through the men's barracks, and no doubt some fruit was purchased from the villagers by them. This, of course, was at once set down as the cause of the outbreak, "human intercourse" and "infected articles of food."

I, however, traced these people to their daily destination, and found that they passed on into the native city of Allahabad, which, like all native cities, was in a very insanitary state. These villagers sold their fruit, vegetables, and grain to the residents, spent the best part of the day in the bazaars, used the latrines as a matter of course, drew water from the wells\* with their brass lotahs and cotton strings, the same, remember, that they used in their infected villages, and yet the inhabitants of the native city remained absolutely free, and even when cholera did appear, two or three months later, the outbreak was not severe.

Instances like this could be quoted indefinitely, and this it is that causes such confusion among theorists; each seeks to fit the facts into his peculiar views, and when they tell against him they are ignored or minimised.

I must allude briefly to the last theory, viz., that cholera is propagated and conveyed by the air. All of you are familiar with the arguments on this point, how ships at sea have been attacked, how the monsoon current carries the disease with it, and so on. Any number of statements have been brought forward to bear on this point, but when they are closely looked into, we find fallacies on all sides.

A very remarkable instance of cholera being (presumably) carried by air occurred at Subathoo and Kasauli, two hill stations near Simla, a few years ago. Subathoo is 4,250 feet above the sea, Kasauli is 6,300 feet high; the stations are 17 miles apart as the crow flies, and separated by deep valleys.

The population of the intervening country was free from cholera, as also were the bazaars and barracks, yet on the same day, the 12th September, after a heavy fall of rain, cholera broke out on the top of each hill simultaneously, lasted for three days, and

\* The new water supply had not then been brought in.

ceased on the same day, the 15th, after which there were no further attacks.

I have merely quoted this instance to show how arguments in favour of any theory can be adduced, and what extraordinary paradoxes are encountered in tracing out epidemic cholera. So far as we at present know, cholera appears to be an "infectious disease," not indeed in the manner the ordinary term infection indicates, as illustrated by small-pox or scarlet fever, but it may be defined as a disease caused by the reception from without of a specific infective material, into previously healthy bodies, under some special influence, possibly of the atmosphere, and that this material so introduced acts as a poison.

We see in India that you cannot light up an epidemic in an uninvaded district, and that, though cases of cholera are constantly being carried by the train into countries free from the disease, it does not, therefore, become epidemic; in fact, that the epidemic progression is quite a separate matter.

Acknowledging, then, that we have much yet to learn of the cause of cholera, what advice are we in a position to give as the result of what we do know?

First, as to quarantine. Quarantine may be summed up as impracticable, useless, and vexatious. At the same time no precautions should be omitted at all ports to examine ships entering, and, in the event of cholera having been on board, the vessel should be disinfected, the sick removed to properly isolated hospitals, and the crew kept apart for at least ten days subsequent to the appearance of the last case.

The provision of properly equipped cholera wards as near as possible to infected localities is, of course, most necessary; but more important, again, should be provision for evacuating infected houses and localities.

Here our Indian experience teaches the immense importance of at once evacuating all infected buildings; and this is, to my mind, an even more important step than removing those who have been attacked to hospital.

My arrangements as regards the troops in London are briefly these. In each barrack a large tent is stored, and, should a case of cholera occur, no matter at what hour, the barrack-room is to be vacated, and the patient removed to the tent.

If any further cases occur, the troops from that barrack will at



once go out into camp and remain under canvas till the epidemic ceases.

Of course such a proceeding would not be possible with a large civil population, but, as far as practicable, evacuation of infected buildings should be adopted.

For fumigation, I consider the simple plan of closing all apertures and burning sulphur freely to be the best and cheapest.

As a disinfectant of dejecta, and to use in sewers, closets, latrines, &c., the crude sulphate of iron in powder is about the simplest and most valuable.

The use of dangerous poisons, such as corrosive sublimate (as lately advocated), by inexperienced persons is much to be deprecated, and would probably cause unpleasant results from careless handling.

As regards the treatment of the disease, I need say little, but will merely remark that, so far as drugs are concerned, the only time they can possibly be of any value is during the premonitory diarrhœa.

I have no hesitation in saying, that a very large proportion of cases that would, without treatment, pass on to cholera, may be checked during the premonitory stage if taken in time.

The great danger of this premonitory diarrhœa is its usually painless nature, and persons suffering from it frequently disregard it till too late.

If cases detected in the early stage are at once placed in the recumbent position and given a full dose of opium combined with an acid astringent, many of them may be checked and will not pass into the algide condition.

Here I may remark that purgatives, especially saline purgatives, should never be taken during cholera epidemics, and also that the treatment of cholera by castor oil, as so highly advocated, is dangerous in the extreme. Should the disease pass on to the condition of true Asiatic cholera, I would point out how utterly useless, if not actually injurious, are all drugs given internally.

When a person is suffering from cholera in the algide stage all the symptoms are caused by the fact that the serum of the blood is draining away through the intestines, and that the patient practically bleeds to death.

In this condition of what possible value can drugs or stimulants given internally be?

Not only are they of no value, but, if not got rid of by purging or vomiting and reaction sets in, they may, by being absorbed, cause the death of the patient. When, therefore, a case of cholera has fully developed, no drugs should be given by the mouth. External frictions, heat to the surface and extremities, and hypodermic injections may be of benefit, but by the mouth nothing should be given but ice to suck or soda water to quench the intolerable thirst. Then, should happily reaction set in, the patient is in the best possible condition for recovery, and the secondary fever and suppression of urine may be treated on common-sense lines.

As you are aware, the injection of saline fluids into the circulation has been frequently tried, but generally with small permanent benefit; the reason of this I believe is the destruction that has been caused to the red corpuscles by the deprivation of serum and the want of oxygen, which reduces the blood of a cholera patient to a dark tarry consistence.

It occurred to me some time ago that it might be possible to save life in the algide stage of cholera by substituting the blood of an animal, whose corpuscles nearest correspond to those of human blood, for the tarry unoxygenated fluid that is unable on mechanical grounds to circulate through the lungs and capillary vessels of a cholera patient.

I am told that of domestic animals the goat nearest approaches these conditions, and my idea, crude no doubt, is to perform direct transfusion from the animal to the patient, using the heart of the animal as the *vis a tergo*.

Briefly, by means of an elastic tube and two glass nozzles, I would propose to connect the carotid of the animal with a vein in the arm of the patient. At the same time I would open a vein in the other arm of the man, and even one in each leg, to allow of the exit of the unoxygenated blood.

The details of such an operation would be very simple, and if the animal were prepared in another room, and the tube passed through an aperture in a screen, the patient need know nothing of it.

Such an experiment as this would, I submit, be perfectly justifiable in cases that were evidently hopeless and *in extremis*, and if any benefit was apparent it would be an encouragement to try it in an earlier stage.

No doubt some of those present here to-night will express their opinion on the possibility of saving life by such means, and should, unhappily, epidemic cholera appear in England next year, we may see direct transfusion such as I have proposed at all events attempted.

The PRESIDENT said that he could speak of the cholera from a good deal of personal experience. In 1846 he tried many methods of treatment during the epidemic, including that by bleeding and saline purges. He remembered how the water theory of the late Dr. Snow was received by the profession with amusement, but he was one of his earliest converts. He had no doubt that the poison was usually taken in in the form of fluid which had been contaminated by the dejecta of a cholera patient. He had seen many proofs of the truth of Dr. Snow's theory in the epidemics in London in the years 1854 and 1865. He asked the author if he did not think that it was of extreme importance to insist on the boiling of water by individuals before consumption.

Dr. MACLEOD (Allahabad) said the incidence of cholera was accidental rather than general, and its progression was also peculiar, being by leaps and starts, and by no means general or uniform, going sometimes forwards and sometimes backwards. He deprecated the use of terms such as "waves," "areas," &c., as utterly fallacious and likely to mislead, and so long as the data which went to make up the general statements were imperfect so long would these statements have little weight.

Dr. W. J. SIMPSON (Calcutta) said : It seems to me that the conflicting views which have arisen regarding cholera in Europe and cholera in India are largely dependent on the different methods of investigation adopted in the respective places. The reason for this difference, and especially for the method adopted in India, is, I think, attributable to the immensity of the country and the paucity of medical men therein who have either the time or inclination to carry out such work. To show what a large amount of time an investigation of this kind requires, I may mention that during the six years I have been in India I have had the opportunity of inquiring into several important outbreaks, and each of these has taken me nearly a month or six weeks' hard work to ascertain with any degree of certainty the cause. It will thus be seen how difficult it is for medical men to investigate the causation of disease, while nearly all their work lies in other directions. In England, where medical men are plentiful, the method adopted is a careful local investigation, *plus* statistics. In India, the method is statistical, *minus*, in the majority of cases, a careful local investigation. So many deaths from cholera are stated to have occurred. These are forwarded to a central office and there compiled with similar statements from other places. A report has to be written and some explanation is required, and accordingly a number of theories are apt to be elaborated. I do not say that all of these theories are wrong ; many of them may be right ; but they are lacking in proof, and no investigation worthy of the name that I am aware of has ever shown that the monsoon or the atmosphere conveyed cholera to a locality where it had not existed previously. On the contrary, the local investigations which have been made by sanitary commissioners and others have demonstrated the fact that cholera has been conveyed from locality to locality by infected persons or infected things, and has spread in the locality where it has been imported chiefly by contaminated water, contaminated milk, con-



taminated food, bad drainage, or some cause not ascertained, but not by the winds. The transportability of cholera by human intercourse in India has been demonstrated over and over again. It was even illustrated in the Hurdwâr Khumb of 1879, notwithstanding Surgeon-Colonel Hamilton's diagram, for the vast majority of the pilgrims came from the Himalayas and from the north, north-west, and west, and very few indeed from Bengal. In the Hurdwâr Khumb of 1891, at which I was present, I do not think more than 500, or at most 1,000, out of nearly a million came from Bengal. Last year I was present at the Ardhodaya Jog in Calcutta, which takes place only once in twenty-seven to thirty years. Cholera broke out in a virulent form, spread in the locality of the festival, and raised the cholera mortality in the city of Calcutta at a season of the year when it is not usual for cholera to appear in an epidemic form. The pilgrims were traced to their homes, and it was found that in several districts they had set up widespread epidemics. While, however, believing that cholera is communicable in the sense of being carried from place to place by infected persons or things, and not by epidemic or pandemic aerial waves, I am not forgetful of the influence of season, which undoubtedly is an important factor, and when we come to its local manifestations I am far from thinking that any exclusive doctrine of propagation of the germ in the soil, and local infection of the air as held by Pettenkofer, or propagation by water as held by others, will explain every local outburst. For instance, epidemics on board crowded emigrant ships, where no layer of soil, wet or dry, or varying in its degree of moisture, exists for the elaboration of the poison, tend to show that local causes appertaining to the soil are not essential for the production of cholera. On the other hand, I have seen in Calcutta, in huts in which there has been an absence of light and air, small explosions of cholera, which have reminded me more of my experience of typhus fever in Scotland, or of diphtheria in England, and which, by no manner of means, could be traced to contaminated water. Two years ago there was an outbreak in Calcutta, which appeared to be directly traceable to sewer emanations. It seems to me, moreover, that exclusive theories regarding the propagation of epidemic disease are not in consonance with our knowledge of bacteriology, though some media may be much more important than others, on account of the different liability to contamination. There can be no doubt, for example, that even in the East the evidence is overwhelming in favour of liquids used by man for drink being the most frequent media or vehicles for the cholera organism, and that the air is not a suitable vehicle unless under special conditions of moisture, as may exist in low-lying land near a marsh or river, or under special conditions of air and light, as in dark or unventilated rooms or huts, or in the forced emanations from contaminated sewers. It is by reason of the peculiar treatment which water receives in India, especially in Bengal, that the disease continues to break out yearly in an epidemic form. Calcutta is not the only town that has had its cholera decreased more than half by a pure water supply. Lahore, Delhi, Nagpore, and Bombay are in the same position. I have travelled a good deal in the East, and everywhere have taken the opportunity of inquiring into the cholera history of the locality visited, and everywhere I have found the water supply to constitute the most important factor in the decrease of cholera. Such is the case in Batavia and Pondicherry, which enjoy comparative immunity by reason of their artesian wells, and similarly in Rangoon, Colombo, Kandy, Singapore, and other towns in the East. My experience leads me to the

opinion that widespread local epidemics are mainly due to contaminated water. I acquired an early lesson to this effect in 1883, when I visited Damietta, in Egypt, to inquire into the cause of the outbreak there, which was the first town affected in Egypt. Damietta is situated on the Nile, which forms a peculiar cup-shaped basin in front of the town during the period that the river is low. The river at this time was particularly low, so as to form almost a stagnant pool, which supplied the inhabitants with their drinking water, and at the same time served as the cesspool for the town. A large fair was held, at which 15,000 persons were present, some of whom came from suspected districts; cholera broke out, and raged with virulence, until suddenly the Nile rose and swept out the polluted basin, and cholera disappeared at once.

Mr. ERNEST HART welcomed cordially the remarks of the last speaker, who had given a series of facts of an affirmative character connecting the cholera outbreaks with a defective water supply. He regarded these valuable statements as a landmark in the history of the investigation of cholera by English epidemiologists in India. Hitherto he had never been able to find any light in the writings from India on this subject; there was nothing but confusion, due, no doubt, in part, to the extraordinary difficulties which beset the investigation of the disease. He also regretted to add that it had come to his knowledge that administrative pressure had been, on more than one occasion, brought to bear on the senders of reports, causing them to arrest any investigation that would tend to incriminate the water supply. Surgeon-Colonel Hamilton had really enumerated but two theories as to the cause of cholera; the water theory and the air theory; for the water theory included both food contamination, human intercourse, and the bacillary theory. As to the monsoon theory, the author had totally abandoned it, for he had given instances where the cholera had travelled against a monsoon blowing steadily at the rate of fifteen miles an hour. Cholera certainly did not travel with the air across Europe; it travelled precisely at the rate at which human beings travelled. When it took fifteen years for travellers to go round the world, cholera took fifteen years also: now that it took five months cholera took the same. When cholera travelled from Astrakan to St. Petersburg, it followed the river only as far as the steamboats went. It was evident that we should brush aside all the imperfect evidence which had been accumulated as to the air theory. Water, and water only, was adequate to explain the epidemic extension of cholera. It was perfectly conceivable that in individual cases a man might inhale it in a close room, the air of which was crowded with cholera germs, but these were only exceptions. A dirty hospital might be full of patients, the cholera cases being mixed with others, and yet not a single nurse or ordinary patient would catch it: this had been proved in the Paris hospitals. He had accurate details with regard to every great epidemic which had occurred for the last thirty years. When it occurred in Italy Rome escaped, owing to its pure water supply. In Naples, when they feared that the cholera was coming, they poured carbolic acid into their cesspools and then found that their drinking water tasted of carbolic; the town suffered severely from the epidemic. Genoa was attacked in spite of its pure water supply, and thus furnished an apparent exception to the rule; till it was discovered that some cholera patients had been passing motions into the small channels which fed the reservoir and thus polluted the supply. The deaths, which had been 100 per diem, fell to three when this source of water supply was cut off. In Spain every kind



of demonstration was furnished of the truth of the water theory. In Madrid, Saragossa, and Mercia epidemics occurred, which proved the case to the hilt. Madrid was a filthy city; in 1855 it was decimated by cholera: in 1885 it had obtained a pure water supply and it escaped. Seville and Xeres also furnished similar examples. Toledo was a filthy city with bad water; when the cholera began the Governor, who was enlightened on these matters, shut up the wells and guarded the river which supplied them, and the city escaped the cholera. Other instances were quoted from the Spanish, Italian, and French epidemics, in which whole populations, living in a cholera-infected district or city, but drinking pure water, were saved from cholera, while the adjacent suburban population, with an impure supply, were decimated. The same history was repeated all through France. It was folly to refer the epidemic to the peculiar influence of certain dry plains, of particular rivers, and of seasons and altitudes as such; all such influences were secondary to the pollution of the water supply. A heavy rainfall, in the case of Damietta (already quoted by Dr. Simpson), washed out the pool which supplied the foul water and thus took away the cholera. But at Marseilles it was found by Professor Marey that, at first, a heavy rainfall increased the cholera, because it flushed into the brooks supplying drinking water the dirty ground and sloping banks where cholera stools had been deposited; but after continuous and protracted heavy rains the cholera at Marseilles ceased, as all the impurities were washed far away. The practical question really worth studying in regard to the causation of cholera was first and always what was the water supply and how to keep it clean. The responsibility of the governing power of India was great in this matter, for experience had shown that cholera had habitually started from there on its epidemic tours through Europe. So long as we neglected to take proper measures to repress cholera in India the responsibility would continue.

Mr. MACNAMARA said he wished to correct one statement of Surgeon-Colonel Hamilton as to the supply of water to the troops located in Fort William. From Dr. Hamilton's paper he understood him to say that the improved water supply to Fort William had commenced in the year 1872, but that the death rate from cholera among these troops had very much decreased from 1863; consequently we should look to some other cause than the water supply to account for the improved health of the soldiers. Mr. Macnamara explained that, although the improved water supply to the town of Calcutta had only commenced in 1873, the troops in Fort William had been given perfectly pure water from and after the year 1863, for in that year the covered tanks constructed by Government outside the fort had been completed, and from this time a constant pure supply of water had been delivered to the troops; whereas, previous to 1863, they had consumed the River Hoogly water, polluted as it was with dead bodies and the drainage of Calcutta. The effect of the change in the water supply had reduced the death rate from cholera amongst the troops in Fort William from 20 per 1,000 to 1 per 1,000, and that in spite of the existence of Asiatic cholera constantly among the vast native population in proximity to Fort William. Mr. Macnamara remarked that, as an Indian medical officer, he wished to state that gentlemen in that Service had written as strongly upon the fact of drinking water being the chief means through which Asiatic cholera was disseminated as it was possible to write, that no one had stronger or more fixed views on this subject than he had, and that, although Mr. E. Hart did not



appear to know it, he had published these views in more works than one on Asiatic cholera during the past twenty-five years.

Mr. Hart was also quite correct as to the part the monsoons took in the dissemination of cholera in India ; so far from being unimportant, the wind had a vast deal to do with spreading the disease : for, as the Marquis of Hastings wrote in 1817, it was by means of the south-west monsoon the large country boats come down and up the Ganges, carrying human beings to the north-west, consisting of a number of natives from the cholera-stricken districts of Lower Bengal. Wherever these fleets of country boats remained, there cholera broke out, and in this way the inhabitants of Patna, Benares, and other large cities on the banks of the Ganges become infected, year after year, with Asiatic cholera. The subject, however was far too wide for him to enter upon at that hour of the evening ; he wished, however, before resuming his seat, to thank Surgeon-Colonel Hamilton for his paper.

Surgeon-Colonel HAMILTON, in reply, said that he was the last to deny the enormous value of a good water supply or the injury caused by bad water, but he held that something must be present in addition to this to explain the phenomena. Numbers of instances were on record where persons had not drunk water at all and yet they had died of cholera. He remarked on the difficulty of proving any one theory to the hilt, and said that if the question of water supply was the sole one, how was it that over and over again British troops, themselves healthy, housed in clean barracks, with an excellent water supply, and yet had suffered severely from cholera, while within a quarter of a mile of them was a very dirty bazaar, the inhabitants of which escaped, though the water they consumed was filthy.

---

*December 12th, 1892.*

## THE IRREGULAR HEART: A CLINICAL STUDY.

By A. ERNEST SANSOM, M.D., F.R.C.P., Lond.

It was my first intention when I was arranging the subject-matter of the Oration which I had the honour of delivering before this Society in 1890 to have included in that clinical sketch a consideration of the various forms of perturbation of the rhythm of the heart. I soon found, however, that the subject would have been much too extensive, and I confined myself to a review of the clinical associations of cases manifesting for protracted periods a morbid acceleration of the heart's action (tachycardia). I have thought that on this occasion it might serve some useful purpose if I reassembled my former data and added to them some

subsequent experiences on the subject of irregularities of the rhythm of the heart—*arrhythmia cordis*. As in the former communication, I desire to present the subject from the clinical point of view—to analyse the evidence afforded by actual cases—to use much caution in making any deductions, and not to enter into abstruse or debatable theorems of pathology.

I may remind you that I analysed seventy-five cases manifesting a long-persistent morbid acceleration of the heart's action—cases which had all been under my own personal observation—that twenty-nine of these showed the undoubted phenomena of Graves's disease and that in a great majority of the remainder the associations were such as to show a close alliance with Graves's disease. I considered that the central morbid manifestation was the tachycardia, the other phenomena of Graves's disease being, as it were, offshoots from the centre.

I now propose to consider the evidence in cases of the irregular heart in a manner similar to that I adopted in the cases of the rapid heart. I have eliminated all instances in which there was reason to suspect valvular or other structural disease of the heart as a protopathic lesion, and have only taken those which manifested a pronounced irregularity for protracted periods. The number of such cases, all under my own observation, has been 47, and I shall divide them in two groups, as I did with the instances of tachycardia—*first group* showing none of the cardinal signs of Graves's disease, 37 cases; *second group*, undoubted cases of Graves's disease, 10 cases.

In the first place it is necessary to outline the *mode of observation*.

1. The radial pulse is to be felt in the usual way and its frequency noted. It is best to observe it for at least four periods of a quarter of a minute, with pauses between the observations. The finger is cognisant of intermission and often of irregularity. Many subtleties, however, may be overlooked, for some of the cardiac pulsations may fail to reach the artery with sufficient force to be distinguished, and the kind and degree of irregularity may be imperfectly appreciated.

2. The frequency of the heart's pulsations should be counted by employment of auscultation, the stethoscope being applied over the portion of the *præcordium* where both sounds are heard with maximum distinctness. A comparison of the heart-rate and



pulse-rate will of course indicate what proportion of the cardiac systoles fails to be observed in the radial. Moreover the observation serves to show with much precision the kind and degree of irregularity—the cardiac cycles may be heard in groups of two or three or more—the well pronounced are distinguished from the less pronounced sounds.

3. The radial pulse is felt by the finger at the same time that the heart-sounds are heard by the stethoscope—it is obvious that this method gives further evidence as to the complete and the feeble or ineffectual systoles and their time-relations.

4. Whilst observations are made by any of these methods the patient is directed to elevate to the utmost one or both arms. The pressure of the blood-column upon the left ventricle may induce a great increase of the rate of cardiac pulsations if the heart be unduly irritable—it may increase or decrease the irregularity. Caution must be used in adopting this expedient. It may cause faintness or other unpleasant symptoms. It is a better method, however, than that of causing the patient to make brisk walking movements.

5. It is by the use of the sphygmograph, however, that we obtain the most precise evidence concerning the kinds and degrees of cardiac irregularity, and it has been employed in the investigation of all the cases that I have to bring before you.

I will now consider the various *forms of irregularity*.

I. *Intermission*.—The heart pauses for a period about equal to that occupied by a single pulsation—there is, as it were, a “skipped” or “dropped” beat. Of course this is a common and well-known form of irregularity, initiated, it would seem, often by trivial causes and having very exceptionally a serious significance. I have found from the graphic evidence (Plate I, Fig. 1) that in some cases the character and the volume of the pulse immediately succeeding the pause do not appreciably differ from those of the other cycles; in others the pause is followed by a much more ample tracing; the output from the ventricle after the rest is increased; in some cases a pulse of less amplitude than the preceding occurs after the pause.

II. *The Alternating Pulse* (Plate I, Fig. 2).—The “*pulsus alternans*” of Traube in its typical form is rare. A more ample is succeeded by a less ample pulse-trace with mathematical precision—there is a rhythmical irregularity in the volume of the pulse. I



have observed this form of irregular pulse in a case of thrombosis of the left middle cerebral artery, the patient manifesting Cheyne-Stokes dyspnoea.

III. *Coupled and Linked Beats*.—It is by no means uncommon to observe that the cardiac pulsations occur in pairs with a pause between. These may be shown in the sphygmogram as pairs of complete tracings having ordinary characters; the pulse has been called the twin-pulse (Plate I, Figs. 1C and 3A). Or there may be groups of three or four complete pulse-tracings with pauses between. When the repeated contraction of the ventricle occurs very soon after the preceding, there being a slight or inappreciable diastolic interval, the second tracing is recorded in the sloping descending line of the first. Thus two, three, or four pulsations may be observed in the descending line (Plate II, Fig. 1). Each of these—which may simulate dicrotic and secondary waves—is a true cardiac cycle occurring in all cases after the closure of the semilunar valves. The difference between the repeated pulsations the commencements of whose upstrokes start from the base-line and those which are manifested in the sloping-down stroke are merely differences of length of diastolic periods. This is explained by Plate II, Fig. 2. It may be readily understood that these rapidly recurring and slightly pronounced systoles may be quite undetected by the finger applied over the radial artery.

IV. *Extreme Arrhythmic Irregularity* (Plate III, Fig. 4).—In some cases the sphygmograms show an extraordinary irregularity—the heart's action seems riotous. There is a veritable “folie du cœur.” There may be in some tracings an extremely dicrotic or a monodicrotic pulse and in others a fairly-pronounced first wave. It would seem probable that the vaso-motor conditions as well as the heart-regulating mechanism are much disturbed, and it is difficult to realise that patients presenting these extraordinary perturbations may nevertheless complain of little or no distress, and may be pursuing their ordinary avocations. Just as a condition of rapid heart may long persist, the rate of pulsations being over 150, without subjective discomfort, so also an extreme state of irregular heart may continue without symptoms, and, it would seem, without danger.

I come now to a consideration of the conditions of disease with which cardiac irregularity may be associated.

1. *Dyspepsia*.—It is well known that intermission or other irregularity may occur in the subjects of dyspepsia or in those whose dietary is at fault. The late Sir Thomas Watson graphically narrated a case of cardiac intermission and irregularity with much subjective distress in which the symptoms disappeared after tea as a beverage was renounced.\* I have observed a like favourable result in a patient who gave up potatoes and sugar as articles of diet. Nevertheless any long-continued irregularity is rare in the ordinary forms of dyspepsia; it seems probable that there must be some predisposition on the part of the patient, who will probably fall within one of the groups hereafter to be considered.

2. *Syphilis*.—In the case of a gentleman, aged 57, who had suffered from primary syphilis about three years before coming under my notice, and who had secondary manifestations, I observed an extreme form of cardiac irregularity. The pulse, counted at the radial, was 72; the heart-pulsations 100; on another occasion pulse 72, heart-beats 120; on another, pulse 68, heart 128. The sphygmogram showed many minute and imperfect traces with others of good amplitude. At this time there was some bronchitis and occasional attacks of dyspnoea occurred. I examined the patient four years afterwards, when there was little or no subjective discomfort, but the cardiac irregularity persisted.

I do not take it as strictly proved that the syphilis was in this case the determining cause of the irregularity; its influence in this direction, however, seemed to me very probable, for no other morbid associations were in evidence, and the patient was not of the emotional type. His attacks of dyspnoea, not amounting to spasmodic asthma, suggested a disturbance of the vagus. It may be remembered that in one of Dr. Bristowe's cases of tachycardia, in another recorded by Drs. Dreschfeld and Maguire, and in one of my own cases there were definite histories of antecedent syphilis. It seems probable that the morbid changes due to syphilis may be such as to induce in some cases a morbid acceleration and in others irregularity of the heart's action.

\* 'The Principles and Practice of Physic,' 4th Ed., vol. ii, p. 257.

3. *Osteo-arthritis*.—I have observed an association of long-persistent irregularity of the heart with osteo-arthritis in six cases. I have found it in the early stages of the disease, and also when the thickenings of and about the joints have been very pronounced. I cannot doubt that, whilst in a majority of those cases of osteo-arthritis in which a disturbance of the nervous mechanism of the heart occurs, this is manifested by a morbid acceleration—as first pointed out by Dr. Spender, of Bath—in a minority the symptom produced is cardiac irregularity.

4. *Disturbances of the Organ of Hearing and Naso-pharyngeal Irritations*.—In this group are included ten of my cases; I must take some of these as examples.

A lady, aged 53, came under my notice complaining of vertical and occipital headache, occasional vomitings, much depression and noises in the ears as of a traction engine. She was deaf as regards the left ear. The heart presented no signs of disease, but irregular action in the mode of coupled beats. The pulse was typically the twin-pulse—the so-called “*pulsus bigeminus*.” On some occasions this coupling would be observed for three or four groups, and then the action would be regular for a time. Subsequent observations showed that the rhythmic irregularity might be replaced by linked beats, the non-rhythmic irregularities that I have already described. The case was observed for a long period; there were occasional so-called bronchial attacks (*vagus storms*?), symptoms of faintness, pain referred to the heart-region and vertigo. Under treatment, chiefly by the bromides, there was much improvement; but more than two years after the first observation, when the patient expressed herself as much improved, a very marked irregularity of the pulse was manifested. There was no satisfactory evidence as to the mode in which the disease commenced. The uncomfortable symptoms were said to have originated twenty-seven years before, when the patient was in India, but she had suffered from no tropical disease.

A second case which affords some further evidence upon our subject was that of a lady, aged 72, sent to me by my colleague, Dr. Woakes, on account of irregularity of the heart. Four years before coming under my observation the patient had been under treatment for intra-nasal disease (*ethmoiditis*), and previously to this the patient said that her ears had been affected. These con-



ditions of disease had greatly improved, and there were only some thickenings of the tissues in the neighbourhood of the middle turbinate bone as a result. There were no signs of organic disease of the heart, the patient was capable of much effort, physical and mental, but she presented signs of very marked irregularity of the heart, which have persisted during a period of nearly four years, during which she has been under observation. Many pains and discomforts were experienced—severe and protracted pain referred to the heart-region, the epigastrium, and the abdomen generally; burning and aching in the throat; occasional attacks of flatulence and of diarrhoea. An unexpected occurrence in the case was the expulsion in the stools of a very large round worm. No others followed in response to treatment. An immediate effect seemed to be a restoration of the regularity of the heart's action. After about three months the irregularity returned. It would seem probable that an irritation of the intestinal mucous membrane was a potent agent of reflex disturbance, but that the patient herself was from predisposition prone to such disturbance of the automatic mechanism of the heart, and that other causes of reflex irritation arose from time to time.

A lesson of practical interest and importance was taught by this case—one which my further experience has abundantly confirmed—that the symptoms of pain and distress complained of by the patient are not correlative with the degree of cardiac irregularity, but that often these are in inverse ratio. At times when severe pain was complained of, whether præcordial or epigastric, the pulse was found to be quite regular, the arterial tension being more prolonged than at the periods when pain was absent. It is important to note that this patient was also the subject of osteo-arthritis of subacute form commencing in phalangeal joints, and not attacking the larger articulations.

A similar association of osteo-arthritis, auditory disturbance, and irregular heart occurred in another patient, a man, aged 72. He had been deaf for two years, and experienced tinnitus aurium—the sound heard being like that of a steam engine. The cardiac irregularity was very great, but was attended by no subjective signs of discomfort.

In four cases there was aural vertigo. In one of these a man, aged 60, the attacks of vertigo and vomiting were very distressing.

The patient was frequently brought home in a cab, unable to stand. There was tinnitus, the sound being sometimes like that of a steam engine, at others that of a tea-kettle. The pulse intermitted frequently (every third or sixth beat), with sometimes irregularities in volume in the intervening pulsations. Much improvement followed a course of ammonium bromide with, subsequently, hydrobromic acid and strychnia, but the most important point was that Dr. Woakes discovered a fibro-cystic growth depending from the middle turbinate bone, after the removal of which the patient manifested great improvement, increasing in strength and bulk, and making a good recovery.

The other three cases were good examples of Menière's disease; the only associated symptoms I need mention other than those usual in the affection were pains down the right arm and in the right breast in one case, pains of the character of pleurodynia on both sides, or another, stiffness of neck and occipital pain, frontal pain and sense of fulness. Pulsations and palpitations were complained of in two cases. In all there was very marked cardiac irregularity; in one case this irregularity was notably increased by movement of the left arm. In one of my cases, a female, aged 47, there was neither tinnitus nor vertigo, but a subjective feeling which was the patient described as "forcing at the back of the nose," and pharyngitis was obvious. Headache was complained of, and there was a sense of coldness in the limbs approaching tingling. The heart's action was very irregular and the patient was conscious of this; she experienced a "stopping of the heart occasionally with a sense of dead weight."

From these data I must conclude that irregularity of the heart is sometimes the result of a reflex from the naso-pharyngeal tract and from the neighbourhood of the auditory mechanism, and that it is often associated with symptoms of disturbance of hearing. It will be interesting to get the experience of aural surgeons on this point. Dr. MacBride says: "Walker Downie and after him Herzog have recorded cases in which irregular action of the heart could be traced to aural affections, such as the presence of wax or polypi, but this symptom is, judging from my own experience and the writings of others, extremely rare."\*

5. *Influenza*.—In four cases I observed marked irregularity of

\* 'Diseases of the Throat, Nose, and Ear.' Pentland, 1892.



the heart which I believed to have been the result of influenza. In one case, a lady, aged 35, a slight attack of influenza occurred two months before the onset of the special symptoms. There were pains in the heart-region, a sense of arrest of the heart's action at intervals, and a feeling of deadness down the left arm occasionally "with pins and needles on recovery." The pulse showed typically the phenomenon of coupled beats—the twin-pulse. Subsequently the rhythmic irregularity disappeared, but intermissions and arrhythmic irregularities occurred. There was a good recovery under treatment by weak continuous galvanic currents from the nape of the neck to the region of the great nerves on each side of the thyroid cartilage and trachea. In another case, a lady, aged 58, who had suffered from influenza four months previously, there were symptoms of discomfort and inordinate pulsation of the heart with very great irregularity. A third case was that of a strong athletic young man, aged 27, who had been ailing since influenza; he was disturbed by trifles, and was much troubled about his heart. The physical signs indicated no disease, but there was marked irregularity.

In the majority of cases in which I have observed the normal rhythm of the heart to be disturbed after influenza this has been in the sense of morbid acceleration—tachycardia. Dr. Althaus has described a case in which he observed a pulse-rate of 100 six months after an attack of influenza; Colley recorded the case of a woman who developed exophthalmos during the attack, and subsequently the other chief phenomena—thyroid enlargement and tachycardia—of Graves's disease were manifested.\* In a minority of cases I have observed a slowing of the heart—bradycardia—as a sequel of influenza. In one case an habitual pulse of 72 was reduced to 48, coincidently with epigastric pain of some hours' duration, the pulse-rate subsequently rising to 92. The cycles of phenomena occurred daily for about a week. Dr. Strange, of Worcester, has recorded a case occurring during the prevalence of epidemic influenza, in which, with sudden feebleness and oppression at the heart, the pulse became extremely irregular and feeble, only 20 pulsations per minute being felt at the wrist. In six hours the regularity of the pulse returned, but subsequently irritative cough and afterwards violent gastric catarrh occurred.

\* Althaus, 'On Influenza.' London, Longmans & Co., 1892, p. 199.



Dr. Strange concluded that a poisonous influence had been exerted upon the vagus centre, all three divisions of the nerve being affected in turn.\* I must conclude from the cases I have observed that the rapid heart, the slow heart, and the irregular heart may all be the legacies of influenza.

6. *Mental Disturbances and the Effects of Severe Nervous Shock.*—Twelve cases came in this category—hypochondriasis, melancholia, groundless fear, “*Metus e causis non æquis*,” and, exceptionally, hallucinations were amongst the associations. It would be interesting to know the experiences of physicians who have large opportunities of observing the insane as to the frequency of irregularities of the heart in such patients. One point I am well assured—that whereas the subject of irregular heart may be for months or years unconscious of his cardiac disturbance, let his attention be overmuch directed to his heart and he begins to suffer from subjective symptoms of pain and discomfort and becomes the cardiac hypochondriac. It is the physician’s duty to turn the patient’s attention away from the disturbed organ and insist that he never count his radial pulse nor listen expectantly for the sounds and signs of the cardiac tumult.

In a considerable number of the cases in this group there have been recurring attacks of severe dyspepsia. In others—and in some of these also—there have been times of more or less dyspnoea, occasionally attacks of considerable severity. I should like to call these periods of heart-tumult and these recurrences of gastro-intestinal and respiratory disturbance by the term of “vagus storms,” if it be not thought that I am wandering from my simple and plain plan of stating clinical facts into the by-ways of conjecture.

It is interesting to note that the only two instances in which it appeared that an acute disease initiated the cardiac arrhythmia were those of pleurisy. One of these, a man aged 45, came to me with signs of pleuritic effusion in the left chest and dry pleural friction over the right upper lobe. He became very hypochondriacal and I sent him on a voyage to the Cape. His recovery was complete so far as the lung-troubles are concerned, but his heart’s action became irregular in extreme degree and so persisted

\* ‘*Brit. Med. Jour.*,’ Sept. 13th, 1890, p. 629.

for two years (Plate III, Fig. 3). The other case was that of a lady, aged 55, who began to suffer from her heart after pleurisy (right side) and manifested a most irregular heart ten years after, there being no evidence of organic disease. In one of my cases in which there was good evidence that a sudden nervous shock initiated the cardiac perturbation the circumstances were very peculiar. A gentleman, aged 41, was suddenly almost suffocated by a portion of a shrimp being stuck in his throat. It was said that he very nearly died and subsequently became very tremulous and depressed. His heart was very irregular. It seemed possible that in this case there was a combination of nervous causes to bring about the result, viz., a sudden mental perturbation and an abrupt shock to the vagus centre from the severe peripheral irritation. It may be a tenable hypothesis that the severe cases of pleuritis may have acted in a somewhat similar mode.

I have only one instance in which it seemed probable that irregularity of the heart might have been induced by physical overstrain. This was in a young man of 18, who had been engaged in a rowing contest about six months previously to my observing him. In this case the pulse was slow, 48 per minute, there was much irregularity in time, and repeated pulsations were frequently manifested in the downstroke of the sphygmogram (Plate III, Fig. 1). I have met with a considerable number of cases of slow pulse—bradycardia—but have not yet observed the conjunction with epilepsy, as noted by Professor Tripier and others, though I am of course convinced of the validity and importance of the observations. My experience, however, entirely coincides with that of Dr. Broadbent in negating the conclusion of Professor Tripier that deviation of the cardiac rhythm with slowing of the pulse-rate does not occur except in epilepsy or under the influence of digitalis.\*

7. *Cases with None of the Foregoing Associations.*—In five cases there were either no notable morbid associations, or these were so complex as to be difficult to classify. In a gentleman, aged 46, of good physique and fair general health, the cardiac irregularity seemed to be associated with no special symptoms or morbid conditions whatever, except palpitations and the feel-

\* Broadbent, 'On the Pulse.' London, Cassell & Co., 1890, p. 111.

ing of irregularity of the heart. The patient was not morbidly introspective. In the case of a lady, aged 41, sent to me by Dr. Allen, of Napier, New Zealand, there was also extreme irregularity, and the only notable associated sign was fugitive œdema especially about ankles and hands. In a clergyman, aged 53, there was fugitive erythema or urticaria and recurring attacks of dyspepsia. In a female of 25, with a sense of fluttering at the heart, there were feelings of extension of the same, as it were, to the throat. In one case, that of a man, aged 53, the associations were more remarkable—first, an ecthymatous eruption, then glycosuria, the pulse during these periods being markedly intermittent, then a restoration of regularity of the heart's action, and ten years afterwards intermittent hæmaturia. This patient is still under observation.

I now come to consider—very briefly in this communication—my second section of cases, viz., those presenting the signs of Graves's disease. These, ten in number, presented in some instances the slighter, in others the most extreme, forms of irregularity of the heart. A few cases manifested the rapid heart on some occasions and the irregular heart on others. I mentioned in my former communication that I had under my care simultaneously two sisters, the subjects of Graves's disease, the one presenting signs of rapidity only of the heart, whilst the other manifested extreme irregularity. The latter, aged 46, had noticed thyroid enlargement for eight years, both lobes being equally increased in size; Stellwag's sign was present, but not von Graefe's, and exophthalmos was slight. A systolic murmur was heard at the base of the heart, but there was no evidence of valvular disease. The pulse-rate varied from 120 to 152, and the irregularity was extreme. During the time when this cardiac tumult was at its height the patient was able to walk about as usual, showing no signs of discomfort, and she slept well. Œdema was noticed in the feet and gradually extended to the thighs and I lost sight of the case. I am afraid there was soon a fatal termination, but the sister made a good recovery. Another case of typical Graves's disease in which the heart presented extreme irregularity was in a man, aged 41. He manifested much tremor of the facial muscles. A female, aged 38, was another typical example. It is in these cases of Graves's disease that I have witnessed the most extreme forms of cardiac irregularity, and yet I have not noticed that the



patients have, except in the one case I have cited, presented more symptoms of distress, nor has the disease gone a more unfavourable course than when the heart's action has been disturbed in the sense of acceleration only.

I have now completed my clinical survey of cases of irregular heart unaccompanied by structural disease of the organ which have come under my own observation. I submit that all forms and degrees of irregularity from the slight to the most pronounced are found in cases of disturbance of the central nervous mechanism of the heart. The associations in cases of irregular heart strikingly resemble those in rapid heart. Both such forms of disturbance of the rhythm are to be found in cases of osteo-arthritis, in those of aural, nasal, and pharyngeal disorder, and especially in Graves's disease. The cases without notable associations might point the lesson that, whilst the central disturbance from which the other affections in Graves's disease are off-shoots brings about in the majority abnormal rapidity of the heart's contractions, in the minority it induces irregularity. So in many instances arrhythmia cordis may be considered a "forme fruste" of Graves's disease. Only it is better to express it that the *ensemble* of the phenomena of Graves's disease is due to an extension from the area of disturbance which is, focally, that portion of the nervous system which is concerned with the regulation of the heart's movements. In all such cases, whether manifesting tachycardia or arrhythmia, outbreaks of dyspnœa or of gastro-intestinal disturbance—vagus storms, as I have termed them—are frequently observed. It seems that, whilst sudden overstrain is more likely to produce a tendency to morbid acceleration, the more chronic forms of mental depression tend to be associated with irregularity. When a sudden shock or intense disease, however, specially involves the afferent fibres of the vagus pronounced cardiac arrhythmia may be the result.

This paper is intended as a review of the clinical evidence only, and not, or only indirectly, to consider any questions of therapeutics. If a future opportunity be accorded me, I hope to deal with the subject of the treatment of the cases of rapid heart and of irregular heart and to ask for your criticism and assistance.

Dr. B. W. RICHARDSON, while recognising fully the extreme clinical value of the paper, said that his attention had been rather directed to

# PLATE I.

## INTERMITTING, ALTERNATING, AND TWIN PULSE.

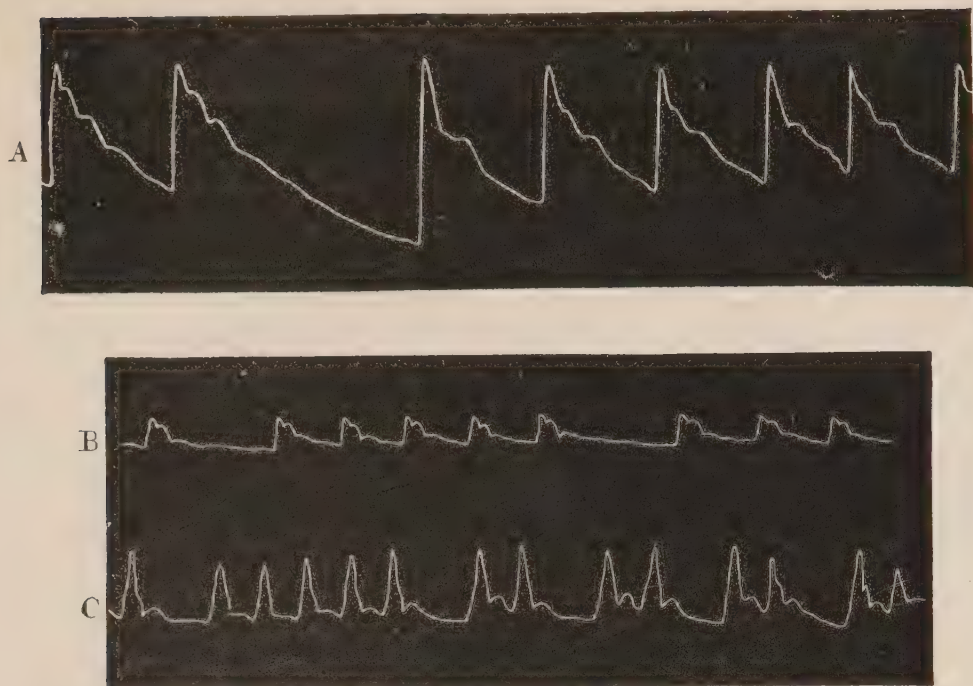


FIG. 1.

Intermission. In A the trace immediately succeeding the skipped beat has a greater amplitude than the average. In B the intermission was found not to influence the amplitude of the individual traces. In C, after some of the intermissions, there were series of twin pulses (coupled beats).

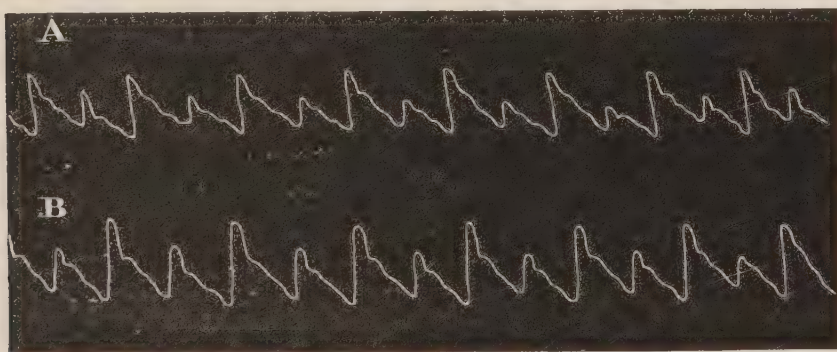


FIG. 2.

The alternating pulse (pulsus alternans), a pulse of larger being invariably followed by one of smaller volume.

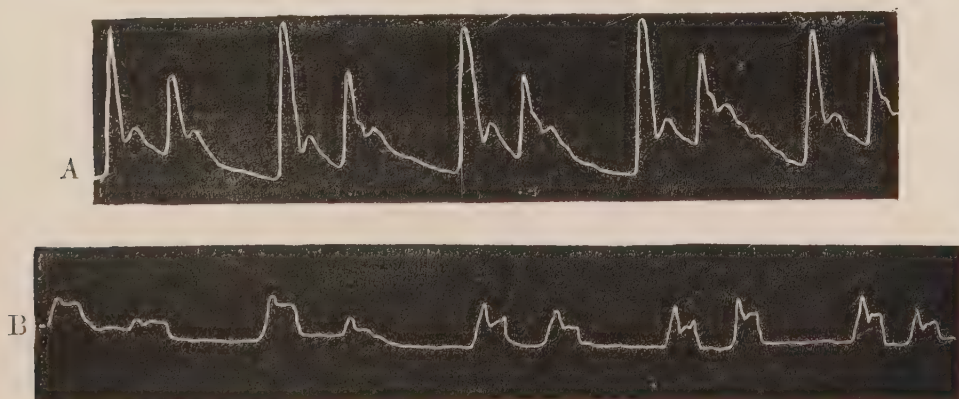


FIG. 3.

The twin pulse (pulsus bigeminus). A, sphygmogram; B, cardiogram.





## PLATE II.

### LINKED PULSES AND THE MECHANISM OF THEIR PRODUCTION.

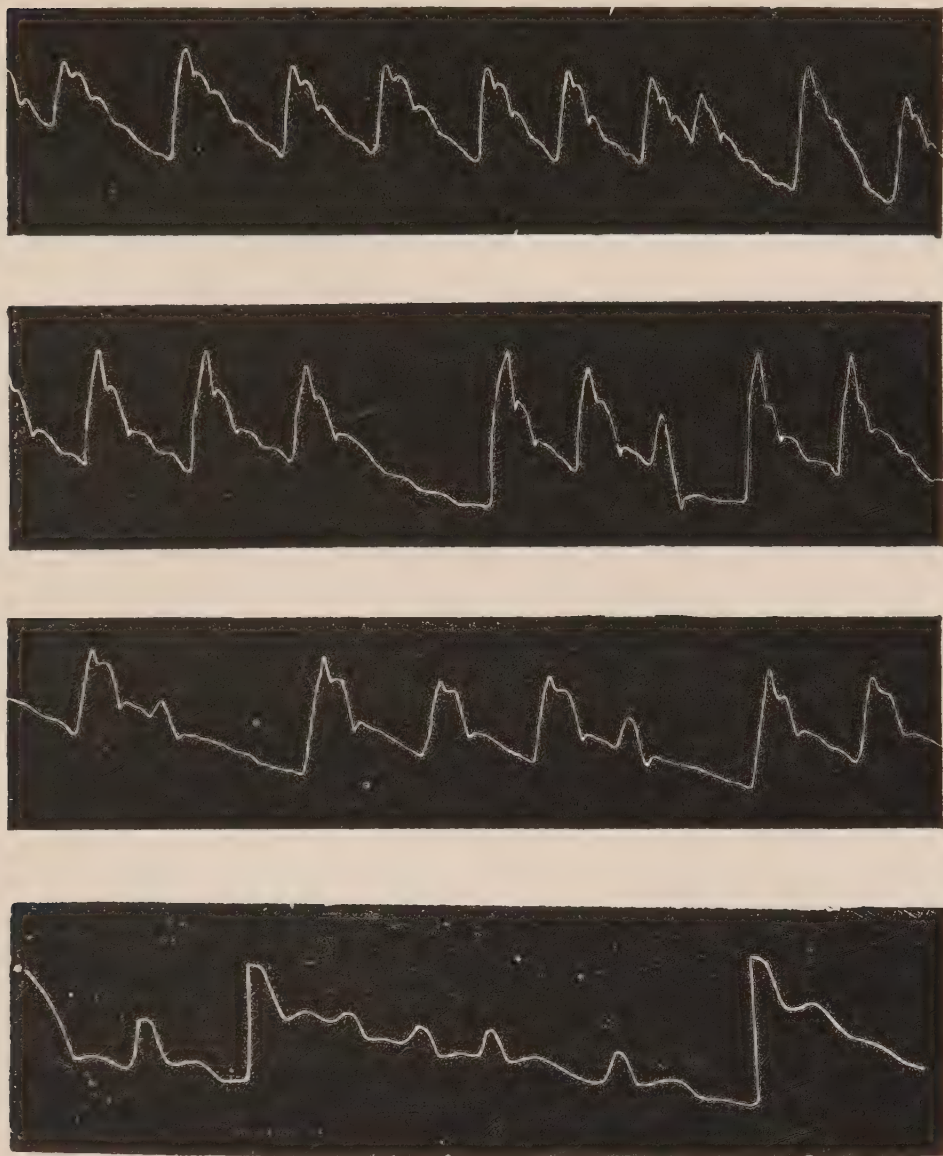


FIG. 1.

Repeated pulsations in the descending line of the sphygmogram (linked beats).  
In the lowest tracing four such pulsations are recorded.

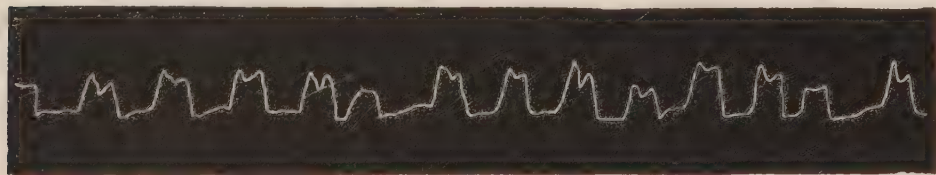


FIG. 2.

Cardiogram illustrating the mechanism of these repeated pulsations. The fourth cycle is followed by a fifth without appreciable diastolic interval, the interval in the last two cycles but one is slightly longer: in each of these cases the pulse would be recorded in the downstroke of the sphygmogram. When, as in Plate I, Fig. 3, the groups presented longer diastolic intervals, the sphygmograms would be in distinct couples, as in Plate I, Fig. 1, C, or Fig. 3, A.



### PLATE III.

#### SOME ETIOLOGICAL GROUPS OF IRREGULAR PULSE.

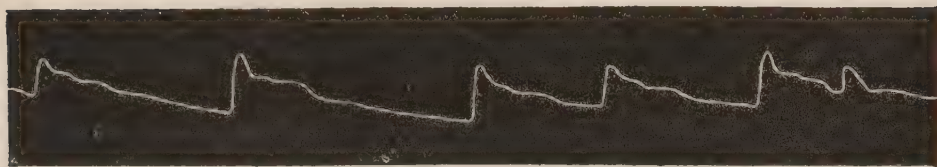


FIG. 1.

Slow pulse (48) in a lad, aged 18, after athletic overstrain.

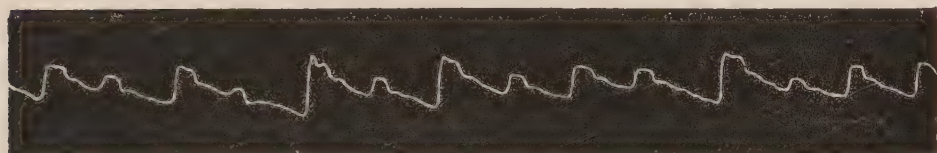


FIG. 2.

Irregular pulse after tropical fever; subjective discomfort at heart.  
Male, aged 47.

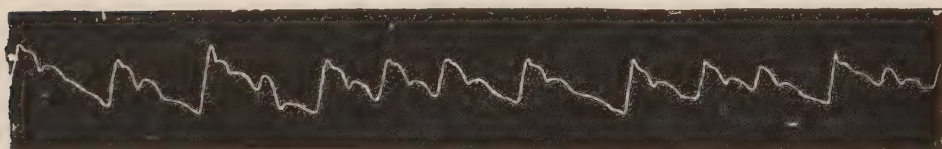


FIG. 3.

Irregular pulse after severe pleuritis with occasional vertigo. Male, aged 45.

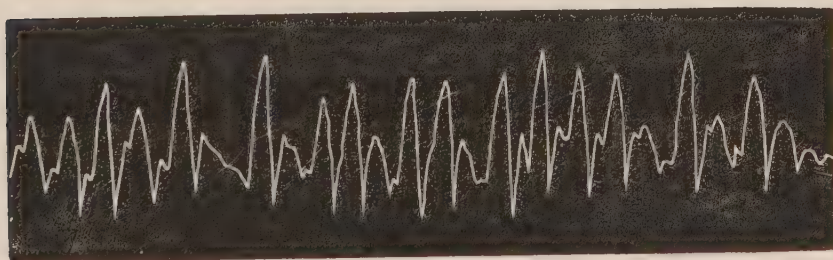
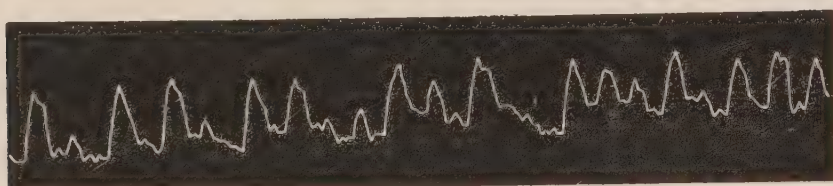


FIG. 4.

Extreme irregularity in two cases of Graves's disease.





simple intermittency, and he had obtained as many as 400 illustrations of this pulse. When intermittency once began, and it usually commenced after 60, it was permanent. It was generally due to failure in the sympathetic nerve supply, though it might depend on irritation of the vagus. As to the effect on the body of irregular cardiac action, it had very little effect provided the patient did not know of it, but it became serious if the patient dwelt on it, and death might then occur from slight causes. There was a modification of the irregular pulse during the course of an acute disease and always to the disadvantage of the patient.

Mr. A. MAUDE said he had met with a rare form of cardiac irregularity in Graves's disease. The beats became feebler and more rapid for a quarter or half a minute, and then increased in force and decreased in rate for an equal time, and the cycle was repeated. It resembled Cheyne-Stokes breathing; it had no reference to respiration, as it continued when the breath was held. He had not found irregularity of the heart such a very common feature in Graves's disease, but had found it the rule in cases of the disease which had manifested itself in old-standing goitre; of these cases he had notes of five, though Graves's disease is ordinarily supposed to occur in goitrous subjects but rarely. Dr. Sansom had referred to the fact that irregular heart sometimes occurs in epilepsy, and Mr. Maude remarked that M. Tripier had shown that epileptics with irregular hearts die suddenly. He once attended a patient who had occasional epileptic fits; he was a drunkard with a neurotic family history, and was also syphilitic. The syphilis may have accounted for the irregularity of his heart which was present. He died suddenly, and no organic change was found to account for his sudden death.

Dr. WOAKES, in response to Dr. Sansom's request, desired to speak as to the condition of the two patients referred to in the paper as having been examined by him. One was a lady of advanced age who was under treatment ten years ago for deafness and nasal affection, who got better, but returned later, when a cleavage of the middle turbinated bone was found, a part of which had become attached to the septum nasi. In his experience heart perturbation or cardialgia was a common consequent on nasal disease. In the second case the patient was suffering from severe tinnitus, with intense vertigo and some deafness. He found a large mass of granulation tissue in the higher region of the nose on the left side, deflecting the septum. He removed the growth with a galvano-cautery, and found a cleavage of the middle turbinated bone, from the interior of which the granulations proceeded. All the conditions referred to above disappeared as the nasal disease recovered under treatment.

Dr. PASTEUR related the case of a woman with irregular heart, in whom the influence of posture was remarkable. The patient was 28 years of age and of nervous temperament. As a girl she had suffered from palpitation—without anæmia. She was married, had one child living, and had had three miscarriages, the last of which, fifteen months before she came under observation, was followed by pelvic cellulitis. Since that time her general health had not been good. In February, 1892, she sought advice, on account of palpitation and occasional stabbing pains in the region of the heart. When first seen the heart presented very typical tachycardia, the beats numbering between 180 and 200 per minute without irregularity. There were no signs of organic disease. At the next visit, a week later, there was extreme irregularity of the heart, with frequent intermissions and a somewhat slower pulse rate. It was observed that as soon as she assumed the recumbent posture the heart almost

suddenly slowed down to 88 per minute and became perfectly regular and free from intermission, but the moment she resumed the upright position all the irregularity forthwith returned. This phenomenon was repeated as often as the patient was made to lie down. A similar condition was noted at two or three subsequent visits, but after six to eight weeks all irregularity disappeared under treatment. There were no objective signs of Graves's disease other than this, but the patient was subject to transient attacks of weakness in the left arm.

Dr. THOROWGOOD remarked that he had seen it stated by a German observer that in cases of pregnancy it would be found that the rate of the pulse was the same whether the patient was standing or lying down. The cause of this was found in the slight amount of hypertrophy of the heart that took place during pregnancy. Dr. Thorowgood also said that when the heart was feeble the effort of raising both arms above the head was very trying to the patient. The test was a very critical one of cardiac failure, and must be applied with caution.

Dr. STEPHEN MACKENZIE referred to the irregular heart which occurred in uræmia, in which there was often a peculiar rhythm, as pointed out by Dr. Wilks. In diphtheria there was a great alteration in frequency of action, together with marked irregularity. He agreed that the prognosis was graver when the patient was cognisant of the malady. He knew of a medical man who had had an irregular heart for twenty years and seemed none the worse for it. He had seen instances where an irregular or intermittent heart had become regular during the course of an acute disease. He quoted a case to show how suddenly rapidity of cardiac action might come on and pass off. In cases of irregularity the trouble often passed off, but in cases of intermittency the condition was generally permanent.

Dr. SOLOMON SMITH said that, although it was necessary for the purpose of the present investigation to separate cases of irregularity occurring in heart disease from those where no disease could be discovered, yet he doubted whether there was any broad line of distinction between the two conditions. Dr. Sansoni had shown how every possible variety of irregularity might occur in the healthy heart in consequence of disturbances quite outside itself, and affecting it only through its nerve supply, and Dr. Smith asked whether, in cases where disease was present, the heart would not be even yet more susceptible to such outside influences, and whether it was not probable that a large proportion of the irregularities met with in actual disease were analogous to those in hearts which were healthy, *i.e.*, due to extrinsic rather than intrinsic causes; at any rate, he thought many observers would bear him out in saying that, in a great number of these cases, the disturbance of rhythm was more amenable to treatment directed to external sources of irritation than to the heart itself. He thought people were perhaps too ready to accept irregularity occurring in an obviously diseased heart as an indication of failure of the muscular apparatus. Not only was it peculiarly apt to be set up by outside causes, but, even when produced by internal difficulties, the irregularity was probably not so much the result of the load of work before the heart being too great, as of its being abnormally distributed. The stimuli in response to which cardiac contraction took place were very complex, and included among them the inherent tendency of heart muscle, with its ganglia, to rhythmic contraction, and the reflex (?) arising from distension of its cavities. Now it was a very striking fact that increase of load before the whole heart tended but little to irregularity,



and that the typical specimens of irregular heart commonly occurred in cases in which the difficulty was placed between the auricle and ventricle in such a manner as to cause the auricle to be filled long before its time, and thus to dislocate from the other rhythmic impulses that most potent one, the distension of the auricle ; and he thought it more reasonable to look on all cases of irregularity as due to disturbance of the nervous impulses leading to contraction than to place some in that category and to consider others, precisely similar in their symptoms, as due to the mere mechanical effect of overload.

Dr. ALLCHIN, in thanking Dr. Sansom for his admirable and useful paper, enquired if the author had noticed among those cases of intermittent or irregular cardiac action due mainly to nervous causes, such as prolonged anxiety, any marked increase in the arterial tension. He had himself met with several cases of this kind, not associated with any structural disease of the heart or kidneys that could be detected. He fully confirmed the view expressed by Dr. Richardson that intermittence in pulse or heart rhythm was serious in proportion to the consciousness of it on the part of the patient.

Dr. SANSOM, in reply, said that one of the lessons inculcated by the cases analysed in his paper was that cardiac irregularity might be perfectly devoid of really dangerous significance. He thought that nasal and aural troubles were amongst the commonest reflexes which started the cardiac derangement. In the diphtheritic cases it was difficult to eliminate the myocarditis which might be present. Though irregularity often ceased with the onset of acute disease, it usually returned after the latter passed away. Intermission was generally persistent, and was serious only to a minority. He had related elsewhere many cases of irregularity coexistent with high tension. He concurred entirely in the view that the irregularities in cases of cardiac disease were ingrafts of a neurotic character on the cardiac lesion, and were not part of the cardiac disease itself. Many cases of mitral stenosis were accompanied by irregularity, due very probably to an interference with the transmission of nerve impulses between the auricle and the ventricle. The irregularity in typhoid fever was probably due to myocarditis. Fatty degeneration of the heart was not usually accompanied by irregularity, contrary to what was once the accepted teaching. Among the deductions from his paper were that cardiac irregularity might co-exist with a sound and good organ, and that irregularity was one of the not infrequent associations of Graves's disease.

---

*January 9th and 23rd, and February 6th, 1893.*

## THE LETTSOMIAN LECTURES: ON SYPHILITIC AFFECTIONS OF THE NERVOUS SYSTEM.

By JOHN S. BRISTOWE, M.D. Lond., LL.D. Edin., F.R.S.

### LECTURE I.

MR. PRESIDENT AND GENTLEMEN,—My first duty is to thank you, and I thank you very sincerely, for the honour you have done me by appointing me your Lettsomian lecturer. But, being profoundly sensible of the fact that the honour entails responsibility, I feel bound to tender an apology for the temerity of which I have been guilty in choosing for my subject a disease of which my experience has been small by comparison with that of some of my auditors, and which has been so well discussed from different points of view by my predecessors—Dr. Broadbent, Dr. Gowers, and our distinguished President. My excuse is that, while on the one hand my choice was made hastily and without due consideration, on the other hand, syphilis is a disease of vast and ever-growing interest. It is one of which I have seen a good deal in its constitutional forms, and to the study of which I have given time and thought, and I propose to treat of it mainly in its relations to the nervous system and from the clinical standpoint, and by the discussion of cases that have come under my own observation and have been interesting or instructive to me. I trust I shall not be deemed irrelevant or tedious if, before entering on my special theme, I venture to place before you a brief statement of the views I entertain with respect to the pathology of syphilis. I do not pretend that these are in any sense novel. Still, it seems to me that it may be convenient and conducive to clearness of apprehension if, in dealing with this complex and Protean disorder, I begin with such a sketch of it as will serve to indicate the lines of thought which will run through my lectures, to define my position in regard to the cases which I shall bring under your notice, and to obviate the

need of subsequent explanatory interpolations. I regard syphilis, as I suppose all educated medical men do at the present day, as a specific infective disease due to the invasion and proliferation of specific living organisms, and as having manifest relations to other specific infective diseases, but more especially, perhaps, to small-pox and the exanthemata, to tuberculosis and leprosy, and to malignant neoplasms such as cancer and sarcoma.

The clinical history of syphilis is briefly as follows:—

1. At the spot on which the disease is inoculated, a hard tubercle is slowly evolved, and almost coetaneously the lymphatic glands next above it become enlarged and indurated, both probably attaining their full development, without any attendant constitutional disturbance, in the course of from six to eight weeks. These phenomena collectively constitute the first stage of the disorder, or primary syphilis. 2. The disease may come to an end at this point; but usually, and in the natural course of events, between six and eight weeks after inoculation, constitutional symptoms almost suddenly declare themselves: a febrile rise of temperature takes place, a rash appears over the skin and fauces, and other tissues and organs not improbably become equivalently affected. This stage of the disease usually varies in duration from a week or two to several months, but occasionally lasts much longer, and may even recur from time to time for several years. It corresponds to the generalisation of the syphilitic poison and of its effects, and is known as secondary syphilis. 3. With the subsidence of this stage the disease itself, in most cases, comes to an end; but not unfrequently—sometimes while the secondary symptoms are yet in progress, sometimes shortly or immediately after their disappearance, sometimes after an interval of many years, and may be during an interval of apparently excellent health—a further series of phenomena manifest themselves. These are the tertiary symptoms, and constitute what is known as tertiary syphilis. The lesions of this stage, unlike the rash of the second stage, are, for the most part, unsymmetrical in their arrangement, originate, as it were, capriciously in different organs, or it may be in only one, and tend to spread locally. They are essentially of the same nature as the primary chancre, and result, for the most part, in grave and irreparable destruction of tissue. 4. Further, syphilis is often transmitted from one or other of the parents, or from both, to the fœtus, either *ab initio*, or in the progress of its



development, or at the time of birth; and thus the child is born infected with the virus of the disease, which, sooner or later, reveals its sinister presence by the evolution of morbid phenomena which correspond to those of the secondary or tertiary stage of acquired syphilis in the adult, but present marked special peculiarities. This is known as congenital syphilis.

I will now briefly compare syphilis severally with small-pox, tuberculosis, and cancer. 1. The resemblances between syphilis and small-pox are obvious, especially if we have regard to small-pox caused by inoculation. In both diseases specific lesions appear at the points of insertion, and are attended with concurrent specific affection of the neighbouring lymphatic glands; in both, after the lapse of fairly definite periods of time, during which, as a general rule, the patient's health remains unimpaired, characteristic constitutional disturbances almost suddenly arise; and in both, after a longer or shorter time (supposing the patient does not meanwhile die), the constitutional symptoms subside and health tends to be restored. Furthermore, in either case, the disease proves to be, in a greater or less degree, self-protective; and in either case it may be imparted by the mother to the foetus. But there are also striking differences; the most important one from my present point of view having relation to the degree and quality of the protection which they severally afford. In the case of small-pox the specific virus at the end of a few weeks disappears absolutely from the system; and not only is there left behind no tendency to relapse or recrudescence of the disease, but also the patient enjoys almost absolute immunity from subsequent attacks on re-exposure to contagion. That syphilis is also in some sense self-protective is proved, not only by experiment and observation, but also by the fact that the secondary stage tends to, and often does, subside spontaneously; but, on the other hand, relapses are not uncommon, and, what is far more remarkable and distinctive, notwithstanding the subsidence of secondary symptoms the specific poison of the disease is apt to lurk in the system, dormant yet potential, and ready, under conditions as yet unknown to us, to become aggressive and to cause serious, though for the most part localised, outbreaks.

2. The relations of syphilis to cancer seem to me quite as striking as those of syphilis to small-pox. We do not at present know whether the cause of cancer is a specific parasitic organism or

whether it is a specific organism evolved out of the living tissues of the sufferer. We know, however, that the disease is specific; that having once appeared in any spot its subsequent behaviour is that of an aggressive parasitic disease; that, in the natural order of events, the lymphatic glands next above the primary tumour become the seat of growths identical with it; and that subsequently the disease becomes generalised and tumours appear from time to time in various parts of the body, until at length the patient succumbs. The differences are, mainly, that we have as yet no sufficient grounds for believing that cancer originates in contagion or is derived from without; that its generalisation is not, so far as we know, attended with sudden febrile disturbance and widespread or symmetrical distribution of specific lesions, but that rather it resembles in its progress the tertiary period of syphilis. Another point of difference is that, while at the point of primary inoculation of syphilis a single chancre only arises, in cancer the disease spreads locally, partly by continuous invasion, partly by the development of outlying nodules. It is interesting to note, however, that tertiary syphilis shows the same tendency to local spread.

3. Now, as to syphilis and tubercle. The lesions in both diseases are what pathologists term "granulomata," and are often remarkably alike in appearance—as, for example, tuberculous and syphilitic tumours of the brain, tuberculous and syphilitic tumours of the lymphatic glands, and lupus and certain cutaneous syphilides. Tubercle, like syphilis, is an inoculable disease, may undergo generalisation, and may remain latent in certain parts of the organism with the liability to unexpected and sudden recrudescence. Its inoculability is proved by experiment on the lower animals and even by the frequent inoculation of Peyer's patches through the swallowing of infected sputum from the lungs. At the same time the disease is spread in the majority of cases by means of infected air or food. That the contagium of tubercle is capable of generalisation, as are the contagia of small-pox and syphilis, is shown in the common production of general miliary tuberculosis by inoculation of the lower animals and by the cases not infrequently met with in the human being in which patients die as of an acute fever in consequence of the nearly simultaneous development of miliary tubercles throughout the organism. But, on the other hand, these are exceptional cases,



and tubercle is, to a large extent, a local disease and remains localised. This tendency to localisation is observed in the case of lupus, which for years may continue to spread over the skin and yet never be followed by tuberculosis of internal organs, in the case of scrofulous glands in the neck and elsewhere, in the case of tubercular peritonitis, and even in the case of ordinary pulmonary phthisis, in each of which the disease may be, and often is, limited to the organ primarily affected. Still, in all such cases, there is the possibility of generalisation; and we often meet with abundant evidence of its supervention. The tendency of tubercle to become quiescent, with the potentiality of assuming an aggressive character after some indefinite period, is not uncommonly illustrated in the course of clinical experience. Thus we may find an apparently effete tubercular mass in the lung become the centre of a swarm of miliary tubercles, and at the same time the source of a fatal outbreak of tubercular meningitis; or an old tubercular kidney, which had long ceased to cause symptoms, the apparent centre of an explosion of tubercular mischief.

It is obvious, then, that, while syphilis in its case-history has close resemblances to various other diseases, it has striking characteristic features of its own which distinguish it from all others, and especially that all its different stages do not correspond to all the different stages of those diseases to which it seems most closely allied. Further, syphilis, like all other specific infectious diseases, retains its specific quality throughout its whole career. In all its stages it is still syphilis, and the specific lesions of its primary, secondary, and tertiary stages, as also those of the congenital form of the disease, are all irritative or inflammatory growths determined by the actual presence in them of the specific living organisms which are its cause. Again, syphilis, like all other such diseases, has a specific proclivity to attack certain tissues and organs in preference to others, a proclivity which is largely determined by relative suitability of soil. And I take it that the differences in this respect which distinguish the secondary and tertiary stages are due simply to modifications in this suitability which have resulted from the protective or modifying influence exerted over the tissues during the former of these stages; and further, that the distinctive features of inherited syphilis are due in different degrees to the operation of the same cause, to differences of vulnerability of the foetal tissues as compared with those



of the adult, and to interference with the developmental changes which are going on in early life.

It is very important for the accurate delimitation of syphilis to bear in mind that it, like other diseases, and indeed more than most, is apt to be complicated and obscured by other disorders and to be followed by sequelæ which, though having no specific connection with it, have been largely—and still are sometimes—regarded as essential parts of it. Thus formerly, and even in my own early days, before antiseptic treatment had been introduced, and when cases of venereal disease were aggregated in special wards, erysipelas, phagedena, and hospital gangrene were frequent accompaniments of primary syphilis; and even now such complications are not uncommon. But there is no doubt that these are not truly syphilitic, but are due simply to the accidental inoculation of the local sores with other pathogenic organisms. Precisely similar accidents are likely to attend the so-called natural cow-pox as it appears in the cow, and hence the local lesions in that affection not uncommonly present much virulence of inflammation—a phenomenon which has misled scientific anti-vaccinationists into the belief that the original cow-pox was a virulent and untamed malady, instead of being, as pure inoculations prove it to be, a mild and comparatively insignificant disorder; and that when similar accidents attend ordinary vaccination they are due to the tendency of cow-pox to revert to its supposed original malignancy. By sequelæ I mean not lesions arising out of syphilis and still harbouring the specific contagium, and capable, therefore, of rekindling or spreading the disease, but morbid conditions due to damage inflicted on regions or tissues by the direct influence of the specific poison, and remaining over, as it were, after the specific element has died out. I refer to such conditions as lardaceous changes in various organs, damage inflicted on the bones of the head or nose, on the eyes and ears, and probably, also, a constitutional weakness of certain tissues, rendering them unduly liable to degenerative and other changes and to the influence of other morbid poisons.

Before completing this introductory part of my lecture, I should like to make a few remarks on the communicability of syphilis. So far as we know, it is only imparted by direct inoculation, and is contagious, therefore, in the restricted meaning of that term. At the same time the contagium of the disease is pre-

sumably an extremely minute living organism, and infests the specific sores and their discharges; and it may be worthy of consideration whether it might not, under certain conditions—as, for example, when the mouth and throat are the seat of disease—be communicable by the breath or through the air. There is no doubt that we meet with cases of constitutional syphilis the source of which appears to be inexplicable. That it is usually imparted by inoculation of the sexual organs is, in a sense, an accident, due largely to the fact that the primary sore is not in all its stages necessarily a cause of serious discomfort to its owner or preventive of sexual congress, and that in the act it becomes freely applied to a delicate, readily inoculable mucous surface. If the local manifestation of cow-pox extended over as long a period as a chancre does, and were attended with as little intensity of inflammation, there is no reason why cow-pox might not be perpetuated as a venereal disease.

Judging from the analogies afforded by other infectious febrile disorders, and from pathological observation, it may be assumed that the syphilitic virus accumulates, not only in the primary sore and indurated glands, but also in all the secondary lesions, including those of the skin and mucous membranes and in the localised growths of the tertiary stage, and that equally from all of these successful inoculations might be made. It may be assumed, also, that during the secondary stage, when the virus is being distributed by the blood, this fluid and tissues, which are not liable to be, or are not, the seats of specific implication, are also infective, though, for obvious reasons, much less potently and much less certainly infective than are the parts wherein the virus has taken root and is growing. During the primary stage, however, and during the period of quiescence which often separates the secondary from the tertiary stage and even during the continuance of tertiary symptoms, the blood and tissues not specifically affected probably remain innocuous. It is, of course, indisputable that, during its primary and secondary stages, syphilis is virulently contagious; and the explanation is obvious. But it is, I believe, largely held that syphilis in the tertiary form is not contagious. This I cannot admit. I am free to acknowledge that syphilis in this period of its career is far less frequently communicated than it is at any other stage; but this is due to the fact that the specific lesions are at this time, as in the first stage, localised, and, for the most



part, so localised (often in internal organs) as to afford little, if any, opportunity for successful inoculation.

I will venture to quote briefly two cases which, though perhaps they may not be generally regarded as absolutely conclusive on the point, seem to me to constitute a weighty indictment against the innocence of tertiary syphilis. The first was a very sad case. A young medical friend of mine, a man of undoubted respectability and honesty, married a young lady of unimpeachable character and wholly free from taint of inherited syphilis. This was three or four and thirty years ago. He was at the time, and had been for some years previously (during which time I had known him), in excellent health. But I was aware that during all this time one of his testicles had been enlarged, and occasionally had troubled him a little. I do not think he ever consulted me about it or that I ever saw it; but he spoke of it occasionally, and attributed it to an injury he had received some years before while riding. His wife, shortly after marriage, became *enceinte*; and when about half the time of pregnancy had been accomplished she became covered with an abundant and quite typical secondary syphilitic rash, which subsided in the course of two or three months under specific treatment. The foetus died *in utero* about a month before the full time, and was born with abundant evidence of syphilitic disease. She remained free from rash until, after a few months, she became pregnant the second time; then at about the corresponding period the symptoms of secondary syphilis recurred, and again subsided under appropriate treatment. The second child was born at term and appeared fairly healthy, but within a week or two developed severe constitutional syphilis, and died therefrom when it was six weeks or two months old. The mother died a few weeks afterwards from phthisis, of which she had first shown symptoms during the latter part of her first pregnancy. She had never previously manifested any phthisical tendency, and all her immediate relatives were and have remained free. I think there can be no doubt that disease localised in the enlarged testicle was the source of contagion. The second case also concerns an old friend of mine. A married man, who has never had any family, inoculated himself accidentally with syphilis. Within less than a year the disease affected his brain, and he became hemiplegic. He recovered, however, in the course of a few months, and has remained free from specific symptoms ever since, and is in a sense



fairly well. About seven years afterwards his wife, who had never shown any signs of such disease previously, consulted me for a characteristic attack of syphilitic roseola and sore throat. From this she recovered quickly under the influence of iodide of potassium and mercury, and she remains well. I may add that the gentleman's disease was not of the genital organs, and that not a shadow of suspicion rests on the wife's character.

In endeavouring to arrange the material which I wish to bring before you in such a manner as to render it interesting, I confess that I have experienced some difficulty; partly because, while syphilitic lesions of the nervous centres fall into several well-marked categories, the symptoms resulting from them are by no means always or even generally so far distinctive as to allow of non-fatal cases being arranged in corresponding groups, and partly also because we cannot always be sure, even when aided by *post-mortem* investigation, that the cases which we are disposed to regard as syphilitic are really syphilitic or conversely. I propose not to meet these difficulties and overcome them, but to evade them; and this I shall do somewhat roughly—first, by dealing for the most part with fatal cases, and considering them as far as possible under the heads of syphilitic disease of vessels, gummata, and inflammatory infiltration of tissues; secondly, by discussing instructive or interesting cases which did not prove fatal, or in which the diagnosis was not verified by *post-mortem* examination, and by interpolating or adding such remarks or comments as the cases themselves suggest.

Syphilitic arterial disease has been for many years past a well-recognised affection. None of the arteries are, so far as I know, exempt from liability, although, according to our present knowledge, some suffer in larger proportion than others. Both the aorta and the pulmonary artery may suffer, as also may any of the systemic branches sufficiently large to have received distinctive names; and there is abundant reason for believing that the smaller and even microscopic arteries are at least as vulnerable as their larger relatives. The syphilitic process may involve the entire thickness of the vascular walls, but it commences in the inner or outer coat, and always mainly implicates one or other or both of these. I suspect that its not infrequent commencement in the inner coat is due to direct inoculation by virus circulating in the blood, and am inclined to agree with Mr. Hutchinson that when the outer

coat suffers primarily it is from the fact that the vessel has become involved in lesions originating in the vicinity. Syphilitic disease causes thickening and irregularity of vessels, with tendency on the one hand to aneurysmal dilatation and rupture, and on the other to stenosis and more or less complete obstruction by thrombosis. The consequences which would naturally follow such conditions are derangement of circulation and impairment of nutrition in the parts to which the diseased vessels are distributed, with on the one hand softening or some equivalent change, and on the other hand hæmorrhage, either from the rupture of a diseased vessel or in connection with diffused degeneration of tissue. It is curious that, although these syphilitic lesions are occasionally widely distributed, they usually occur in limited districts, the vessels elsewhere remaining wholly or for the most part healthy. Before proceeding to quote characteristic cases of the association of cerebral symptoms with syphilitic lesions of cerebral arteries, I will venture to bring before you three cases in which no doubt there were either central nervous lesions or symptoms referable to the nervous centres, but in which these were of secondary if not trivial importance. Their main interest, indeed, depends on the fact that they furnish admirable illustrations of the characters and consequences of advanced syphilitic arterial disease.

The first of these was brought before the Pathological Society by Dr. Walter Edmunds on May 3rd, 1892. The patient was a man aged 36, who came under Sir William MacCormac's care on account of an aneurysm on the right side of the neck, which had been rapidly increasing in size. He had a few days before admission been seized with a sudden attack of faintness, and after admission the mere handling of the tumour frequently brought on similar attacks. Owing to this circumstance, to the belief that his arteries were extensively diseased (for no trace of pulsation could be felt in any of the arteries of either upper extremity, and there was a loud basic systolic murmur), and to the fact also that the patient was exceedingly ill, it was decided not to attempt any operative procedure. I was consulted in the case and concurred in this decision. The patient had had well marked syphilis twelve years previously. He died comatose twelve days after admission. The heart was large and the pericardium adherent by easily broken-down adhesions. The valves were healthy. A gumma about 2 inches in diameter projected from the groove between the



right auricular appendage and the pulmonary artery. The whole of the thoracic aorta, including the arch, was enormously thickened, all the coats being involved, but the outer coat much more so than either of the others. In places they were collectively more than eight times as thick as natural. The thickening extended along the innominate and right subclavian, the latter of which would only admit of the passage of a bullet probe. The right common carotid was healthy, but the right internal had springing from it near its origin a globular aneurysm about  $2\frac{1}{2}$  inches in diameter. The left common carotid was diseased and only admitted a bullet probe, and the left subclavian about an inch from the aorta ended in an impervious fibrous cord. The cerebral and abdominal arteries were all healthy, and no other visceral syphilitic lesion was discovered.

The second case was exhibited before the same Society and on the same evening by Dr. Herbert Hawkins. It was that of a girl, aged 11, who was admitted into St. Thomas's Hospital suffering, it was supposed, from acute nephritis. After experiencing one or two short attacks of illness, probably due to infarction of the lungs, she was observed on February 7th to have swollen and cold legs and a few days later swelling of the face. She was admitted on the 20th, being at this time extremely ill. She had general dropsy, was passing very little urine, which contained a small quantity of albumen but no casts, and there was evidence of pulmonary congestion. Subsequently she passed a little blood on one occasion with her urine, which continued for a time to be very scanty. But during the last four days of her life it became fairly abundant and ceased to be albuminous; the lower part of her lungs became solid, her pulse of high tension, she presented the Cheyne-Stokes breathing, and died (apparently from uræmic poisoning) on March 2nd. At the *post-mortem* examination gross disease was found in nearly all the arteries of the body. The first part of the aorta was studded with grey translucent spots and patches, from the size of a split pea to that of half a crown, and for a length of 3 inches, immediately above the bifurcation, the thickening was so great that the channel barely admitted a bullet probe. Similar patches were observed in the common carotids and subclavians, in the left internal carotid as it entered the cranial cavity, and in the right internal carotid involving the origin of the middle cerebral. There was similar disease through-



out the pulmonary arterial system. The renal arteries towards their entrance into the kidneys were completely obstructed by clots, which were old and white at the periphery, but red and comparatively recent centrally. There was an old infarct in one kidney, but neither organ showed any evidence of nephritis. The liver presented a patch of peritonitic thickening and was rather large. The spleen was large and firm and its capsule thick and covered with old adhesions. The lower and back parts of both lungs were consolidated and contained large infarcts. There was hæmorrhage into each lateral lobe of the cerebellum, the clot in each case being the size of a hazel nut. No history of syphilis was elicited and the teeth were quite normal. There can be no doubt, however, that the lesions in this case were the result of congenital syphilis.

The next case is one which I exhibited before the Pathological Society on April 1st, 1856, as an example of aneurysmal dilatation of the coronary arteries of the heart. The patient was a sailor, aged 22, who was admitted to hospital for what was supposed to be fever, to which paraplegia succeeded, and who died at the end of three months apparently from exhaustion. The case was not under my care, and I can give no further clinical details concerning it. The pericardium was healthy. The heart was somewhat enlarged; and in the course of the arteries ramifying over its surface were observed a number of nodules, isolated and in strings, and individually from the size of a pea downwards, the nature of which at first sight was not very apparent. Dissection, however, showed that they were aneurysmal dilatations of the trunks and branches of the coronary arteries. In some situations the vessels presented an irregular sacculated condition, involving them from  $\frac{1}{2}$  inch to 2 inches of their length, and in some instances continuous series of vessels were affected. The parietes of the dilated parts were thick, firm, and apparently consisting of dense fibroid tissue. Some were empty, others were filled with adherent, tough, buff-coloured clots. The muscular walls of the heart and its valves were healthy, as also was the arterial system generally. There were several rather large patches of pulmonary apoplexy in the right lung. The liver and spleen were congested. The kidneys were of normal size, but the cortical substance of each was thickly studded with buff-coloured patches and dark red-coloured tracts which seem to have resulted from hæmorrhage occurring at dif-

ferent times. I have described the brain and spinal cord as healthy.

I am not aware that at this time (thirty-six years ago) any suspicion was entertained that the arterial system was apt to suffer in the course of constitutional syphilis; neither was it then the custom to examine the texture of the nervous centres so critically as has been done of recent years. Considering, however, that the patient died paraplegic, I have now little doubt that morbid changes might have been discovered in the cord had it been treated and examined by modern methods. No history of syphilis is recorded in this case; but there is also no reason to believe that any suspicion of syphilis was entertained while the patient was under treatment or that he was ever questioned on the subject. But I have for many years now regarded the specimen which I bring before you as a typical example of syphilitic disease of the coronary arteries, and the case as one in which paraplegia was due to similar disease of the smaller arteries distributed to the cord.

In the following year, November 3rd, 1857, Dr. Peacock and I showed a case at the Pathological Society which seems to me to be of singular interest. It was that of a pitman, aged 35, who had been admitted under Mr. Le Gros Clark's care for hydrocele. This was tapped, and the testicle was then found to be much enlarged. Three or four days after admission he began to lose the use of his right arm, and subsequently his speech became affected. It was then ascertained that six months before admission he had had a fit, followed by paralysis and failure of sight, from the effects of which he had in great measure recovered. The patient was now transferred to one of Dr. Peacock's beds. At this time he appeared to be intelligent, but his speech was imperfect, his mouth was drawn to the left, and he had almost completely lost the use of his right arm, but he could move his legs freely. There was no anæsthesia. The pupils were widely dilated, more especially that of the left eye, and scarcely responded to light. He could swallow solids but not fluids. He improved a little for a few days. But twenty days after admission into the medical ward he had a fit and became comatose, in which condition he died two days later. The calvaria and dura mater were healthy, the subarachnoid fluid scanty. The large veins ramifying over the surface of the brain were distended with blood, but there was little capillary congestion.



The substance of the brain was everywhere of normal consistence and colour. The ventricles contained but little fluid. The basilar artery in nearly its whole length was white, opaque, and solid; its canal was occupied by a cylindrical body, the anterior extremity of which was rounded, while the posterior was bifid and formed a nipple-like projection into each vertebral artery. This body was hollow and contained a little colourless fluid, but its parietes were tough and greyish and adherent to the arterial walls. The adhesions, however, were neither dense nor universal; and it seemed possible that there was space for a little blood to pass between them. The cerebral portions of the internal carotids had not more than half their normal diameter; and both they and their branches for a length of half an inch were blocked up by solid adherent coagula of old formation. All the other arteries at the base were healthy and contained either fluid blood or soft coagulum. The heart was large, but its valves were healthy. There was a small hydrocele on the right side, and the corresponding testicle was in a state of chronic inflammation. All the other viscera were healthy.

This case is remarkable from the fact that at the necropsy all the main arteries at the base of the brain appeared to be completely obstructed by old coagula; and the puzzle as to how the cerebral circulation and the normal consistence of the brain had been maintained seemed insoluble. I can only suppose that the brain had been imperfectly supplied through the agency of the partially obstructed basilar and the circle of Willis, and that his final seizure was connected with some increase of obstruction taking place here at the time of his last fit. It seems odd to me now that I seem to have had no suspicion that the arterial disease was syphilitic either when the case was exhibited, or a year later, when I read before the same Society a paper entitled "An Analysis of Seven Cases of Obstruction of the Cerebral Arteries," in which I not only included this case, but quoted two others with a definite history of syphilis, and ventured to suggest of them that the arterial disease was syphilitic. It is true that no history of syphilis was obtained, and obviously no suspicion of it was excited by the condition of the testis. I have no doubt now that the affections of the testis and of the arteries were both syphilitic.

One of my two supposed syphilitic cases just alluded to is narrated at some length in the tenth volume of the 'Transactions of



the Pathological Society,' p. 21. The patient was a steam-packet engineer who was admitted to hospital on January 4th, 1859. He had had three fits: the first some years previously, the second 18 months before admission, and the last eleven days. He had continued to follow his employment (although after the second fit he had become liable to headache and vertigo) until the occurrence of the third, which was followed by partial left hemiplegia and indistinctness of speech. On admission he was still suffering from hemiplegia and thickness of utterance, and appeared imbecile. While under treatment the imbecility increased, and he became troublesome and spiteful, and on one occasion threw his fæces at the Sister. On February 25th he passed into a comatose condition and remained thus for twenty-seven hours, when he died. No history of syphilis was noted. At the *post-mortem* examination, however, the scars of ulcerated buboes in the groins were observed. On removing the calvaria the dura mater over the greater part of the upper and lateral aspect of the left anterior cerebral lobe was found to be thickened, rough, and a little congested, the corresponding surface of bone being rough and softened. The surface of the brain generally was healthy, though a little congested, but the dura mater in the region before indicated, and in an area of about 8 square inches, was firmly adherent to it by means of a layer of fibroid tissue in which, and partly in the subjacent brain, were embedded two or three tough, opaque, white, fibrous masses from the size of a hazel-nut downwards; the grey matter corresponding to the adhesions was softer than natural, and in places had wholly disappeared; in the anterior part of the left corpus striatum was an imperfect cyst, apparently of hæmorrhagic origin, and about as large as a hazel-nut; the anterior half of the right corpus striatum was congested and much softened; the rest of the brain was fairly healthy; the left internal carotid and its branches for about an inch were buff-coloured, and looked atheromatous, and on slitting them up they were found to be filled with adherent cylinders of tough, old coagulum; the other vessels were healthy; the heart and lungs were normal; the liver was attached to the diaphragm by numerous old adhesions which corresponded for the most part to deep fissures in the surface of the organ, and these in their turn corresponded to cicatrix-like tracts in the substance of the liver, embedded in some of which were small, knotty tumours; all the other organs were healthy.

I remarked of the case that "it was exhibited chiefly in reference to syphilitic disease of internal organs which had been several times brought under the notice of the Society by Dr. Wilks and other observers. The kind of deposit supposed to result from the syphilitic poison was recognised both in the liver and in the brain." Later, in the same volume, when I again referred to this case amongst my group of seven, I spoke of it and one other case, which I shall not now quote, in the following words:—"In two of the cases the patients had suffered from well-marked syphilis. Is it possible that the secondary cachexia of this disease is capable of producing the condition of cerebral arteries under consideration?" I may add that even at this time what are now known as gummata of internal organs were by no means generally regarded as syphilitic, and that, so far as I know, syphilitic disease of arteries was not recognised. Indeed, I may point out, as showing how little this latter lesion was suspected of having any association with syphilis, that even our President, in his excellent article on constitutional syphilis, which appeared seven years later in "Reynolds's System of Medicine," makes no allusion whatever to the subject.

In the sixteenth volume of the Transactions I published another case. J. C——, a carpenter, aged 27, came under my care on May 2nd, 1863. He had contracted syphilis four years previously and had had secondary eruptions at intervals since. He had been attacked four days before admission with pain in the head and giddiness without loss of consciousness, and at the same time lost power in the right arm and in the legs. On admission he was quite sensible, but his speech was imperfect. He complained of giddiness and of aching in the shoulders. His right arm was powerless and his mouth was drawn to the left. His legs appeared fairly normal. There was no anæsthesia or affection of the special senses, and his pupils acted to light. He became drowsy some days after admission, but there was no material change in his condition until the morning of the 16th, when, after complaining of pain in his head, he suddenly became insensible. He never recovered complete consciousness, but would now and then respond by opening his left eye when shaken or spoken to loudly. The pupils were equal and acted to light. The left arm lay limp and entirely without motion, but all the other limbs, including the previously paralysed right arm, presented occasional slight convulsive move-



ments. During the last few days of life he was completely comatose, his convulsive movements ceased, and his eyes became divergent. He died on May 28th. The skull and dura mater were healthy. The arachnoid was somewhat opaque and there was a little turbid fluid in its cavity. The pia mater was slightly congested. The arteries at the base were healthy with the exception of the right middle cerebral, which was completely obstructed by a firm decolorised clot. The middle lobe of the right cerebral hemisphere was softened and the grey matter in parts congested, in parts paler than usual, and yellowish. The left corpus striatum presented a cavity about  $\frac{1}{3}$  inch in diameter, containing turbid fluid and bounded by vascular walls. The heart was healthy, the lungs pneumonic in their lower lobes. The capsules of the liver and spleen were thickened in patches, but otherwise these organs were normal. The kidneys were small, and presented a few irregular depressions on the surface. I remarked of the case that it was interesting as furnishing an example of obstruction by clot of one of the middle cerebral arteries without any discoverable source of embolism, and from the fact that the patient had, to within a short time of his last illness, suffered from syphilis, which I had already shown to be a not improbable cause of such obstruction. I remarked, further, that the obstruction of the right middle cerebral must have been the cause of the sudden aggravation of symptoms, with paralysis of the left arm, which took place twelve days before death; and that his original attack of right hemiplegia must be associated with the presence of the cyst discovered in the left corpus striatum. It seemed to me doubtful whether this had resulted from hæmorrhage or softening, but I thought it probable that in either case it had been determined by disease of some of the minute terminal branches of the left middle cerebral distributed to the affected region.

Since the case just narrated was published, now nearly thirty years ago, I have naturally seen many other cases of syphilitic disease of the cerebral arteries, in some of which the arteries were alone implicated, in more of which the arterial disease was complicated with gummata, and amongst them two cases under my care, which were published as Cases 1 and 2 by Dr. Greenfield in his communication to the discussion on visceral syphilis recorded in the 'Transactions of the Pathological Society' for 1877. But there is naturally a certain amount of sameness in cases of cerebral



arterial disease, and I shall content myself, therefore, with devoting the remainder of this lecture to the consideration of a case or two in which the disease was limited to the basilar or its branches, and in which the pons or neighbouring parts had undergone softening. I published a paper on this subject in the 'Lancet' of July 7th, 1883. The cases quoted therein were six in number, of which only two were certainly syphilitic; but I strongly suspected, though I had no proof, that two or three of the others were of the same nature.

The first undoubtedly syphilitic case was that of a young man aged 33 who had contracted a chancre exactly six months before his death, and who was suffering from secondary symptoms in the form of psoriasis when his fatal illness seized him. Excepting for his secondary symptoms he had enjoyed good general health until within three or four weeks of his admission. He was then attacked with occipital headache, which was followed in the course of two days by frequent vomiting. He was admitted on April 16th, 1872, at which time he was still suffering from headache and presented abundant traces of his syphilitic rash. Two days after admission he began to ramble a little and to be troublesome; and the next morning it was noticed that his pupils were contracted, his conjunctivæ congested, and his left eyelid drooped slightly. Five days later, when he awoke in the morning, he was found to be hemiplegic on the left side; and after another four days it was noticed not only that the left lid still drooped a little, but also that there was paralysis of all the muscles of the right eye, excepting the superior oblique. Meanwhile the patient was becoming more and more drowsy and stupid; and he died the fourth day after the paralysis of the muscles of the right eye had declared itself—namely, on May 2nd. Notwithstanding the fact that the first evidence of brain disease had shown itself in less than five months after he had contracted syphilis, it was naturally assumed that the patient was suffering from cerebral syphilis, and he was treated accordingly. Further, it was not difficult to speculate from the symptoms as to whereabouts the cerebral lesion would be found. The left-sided hemiplegia pointed to involvement of the motor tract issuing from the right hemisphere of the brain; and that the part directly implicated was the right crus cerebri, or the contiguous portion of the pons, had been foreshadowed by the slight ptosis observed in the left upper eyelid. This surmise was confirmed

when a few days later total paralysis of the right 3rd nerve was added to the other phenomena. At the *post-mortem* examination there was found syphilitic thickening, with obstruction by clot, of the right posterior cerebral artery, and consequent softening, with more or less disintegration in patches of the right crus cerebri and of some of the neighbouring parts to which this artery is distributed. The paralysis of the right 6th was no doubt due to thickening of the membranes observed at the base of the brain; and the very partial paralysis of the left 3rd was connected with some thickening of its trunk. It may be assumed that the patient's early and comparatively vague cerebral symptoms were caused by the disease of the arterial walls and slight associated inflammation of the membranes in the neighbourhood, that the hemiplegia took place when the channel of the posterior cerebral became suddenly occluded, and that the right 3rd nerve got involved as the area of softening extended. Beyond considerable excess of fluid in the ventricles there was no further sign of disease in the head; the abdominal and thoracic organs were all healthy.

The second case was that of a young man aged 27, who had had primary syphilis about two years before the symptoms of cerebral disease arose. He was admitted under my care on December 24th, 1881. For three weeks he had complained of pain in the frontal region, which had become more severe during the last two days. There had been no sickness. During the night of the 23rd he was seized with almost complete hemiplegia of the left side, associated with rigidity of the arm and leg, inability to speak, and well-marked paralysis of the 3rd nerve on the same side. On admission he was still partially unconscious; the pupils were dilated and equal; there was external strabismus of the left eye and ptosis of the left eyelid; the mouth was drawn slightly to the right, the tongue was protruded markedly to the left; he was unable to speak, but seemed to understand what was said to him and indicated that he suffered from pain in the frontal region; no difficulty of swallowing; the left arm was flexed at the elbow and lay across the chest, and the hand was closed; the whole limb was rigid and completely paralysed; the left leg also was rigid but extended, and he retained slight power over the movement of the toes; the patellar reflex was brisk on both sides, but more so on the left; no ankle clonus; plantar reflex almost absent on the left side; left leg colder than the right; no anæsthesia. The next day he had attacks of partial



unconsciousness, during which the head and eyes were strongly turned to the left. On the following day he manifested some return of the power of articulation, and was evidently not aphasic. All signs of paralysis of the left 3rd, portio dura, and hypoglossal had disappeared, and the paralysed arm and leg had become limp. Three days later the urine, which from the beginning had to be drawn off, had become alkaline and offensive, and on that day his temperature reached  $103.4^{\circ}$ . After this the pulse increased in frequency, the temperature rose irregularly, and on one occasion he had a prolonged rigor; there was a slight return of the paralysis of the left 3rd, facial, and hypoglossal; and the left arm and leg again became rigid. He then passed into a state of coma, and at the time of death (namely, noon January 4th, 1882) his temperature had risen to  $109.2^{\circ}$ . The vessels of the pia mater were injected, but no trace of meningitis was discoverable. The posterior cerebral and some of the smaller arteries in the neighbourhood were much thickened, yellowish, and opaque, apparently the seat of syphilitic disease; but the other and larger vessels were healthy. There was slight comparative softening of the left temporo-sphenoidal lobe, and the pons, which was somewhat smooth, was soft and semi-fluctuating. On incising this part, an irregular patch of broken-down brain substance was found to occupy the greater part of its right half, being separated from the surface and from the surrounding healthy substance by a zone of congested and apparently inflamed tissue. The rest of the brain substance was normal. No other important signs of disease were found. The *post-mortem* examination does not clearly explain the concurrence of left hemiplegia and slight paralysis of the left 3rd; but, on the other hand, the softening of the right half of the pons accounts both for the left hemiplegia and the deviation of the eyes and head to the left. It is interesting in this case that, while some of the larger arteries at the base were diseased, they were not obstructed and that, while the pons was the chief seat of softening, the basilar artery was healthy. I suspect that in this case, as in some of the cases I have quoted, where there was manifest syphilitic disease of arteries at the base associated sometimes with obstruction, and where patches of softening were found in regions with which these diseased arteries had no connection, the softening was due to obstructive disease of the smaller vessels ramifying in the softened district.



## LECTURE II.

MR. PRESIDENT AND GENTLEMEN,—Before bringing my remarks on syphilitic disease of the arteries of the brain to a conclusion, I wish to place before you two cases, as I believe, of this affection. In one case the symptoms resembled those of general paralysis of the insane, and in the other the main symptoms were those of chronic dementia. A carman, aged 33, came under my care on November 11th, 1889. His family and personal history were both good, and he did not acknowledge that he had at any time had syphilis. His illness began in July with pain referred to the back of the head. This soon became constant, though liable to exacerbations, and associated with sleeplessness and low spirits. In the course of a month or two he began to find some difficulty in writing, and a little later tremulousness of the hands and lips was observed, his speech became affected, and his memory began to fail. On admission he was still suffering from all the symptoms above enumerated. He answered questions sensibly; but his speech was slow and hesitating, and attended with slight tremors of the lips, specially noticeable at the beginning of words. The tongue also was slightly tremulous. His pupils were unequal, the right being the larger, but acted to light and accommodation. There was no strabismus or nystagmus, and the fundi were normal. His hands were markedly tremulous. He wrote slowly and hesitatingly and his handwriting was shaky. He could walk without difficulty, could stand with eyes shut, and turn readily. The tendon and plantar reflexes were very brisk. There was no paralysis and no impairment of the senses, and the abdominal and thoracic viscera appeared all to be healthy. For a few weeks there was no material change in the patient's condition. Early in December, however, he became more and more stupid and drowsy, restless (mainly at night), frequently attempting to get out of bed for no obvious purpose, and needing a special attendant; his speech, still attended with tremor of the lips, became unintelligible, his muscular strength failed generally, and he passed his evacuations without control. It was thought at this time that the right side generally was a little weaker than the left, and that the mouth was drawn to the left. There was knee-clonus on the right side. He continued for a couple of weeks to get worse

physically and mentally, passed into a state of almost complete coma, and it was thought that the fatal end was approaching. But at the end of this time he began, almost imperceptibly, to mend, and in the course of another week was better and brighter than I had ever seen him. The improvement was maintained, and on January 20th he was discharged in much better health than when he was admitted. Indeed, but for slight hesitation in his speech, tremors of lips and hands, and inequality of pupils, he appeared to be a healthy man. All traces of the suspected right-sided paralysis had disappeared. It may be added that during his residence in hospital his temperature ranged for the most part between  $99.2^{\circ}$  and  $101.4^{\circ}$ , and that he was treated with iodide of potassium and mercury.

The man was readmitted on March 15th, 1890. It appeared that for three weeks after leaving the hospital he had remained fairly well, but that then his headache returned, he became dull, taciturn, and stupid, and took to his bed. On admission he was semi-comatose, his face was expressionless, he took little or no notice of what was going on, and answered questions only in monosyllables; his pupils were still unequal (the left being now the larger) but acted to light and accommodation, and the fundi were normal; his lips trembled in speech, and his hands were tremulous. He could stand, and even walk tottering with assistance; the tendon reflexes were extremely brisk. He remained under treatment for eleven days, during which time he got into the same state as he had been in when at his worst in his previous illness, but towards the end of this period it was observed that his left arm and leg were becoming decidedly weaker than their fellows, and that his eyes diverged. His temperature during this time varied between  $95.4^{\circ}$  and  $98^{\circ}$ . On the 26th he passed into a state of complete coma, and his temperature (which had been  $99.8^{\circ}$  the evening before) rose from  $102^{\circ}$  in the early morning to  $106^{\circ}$  at 4 P.M., shortly after which he died.

The resemblance of this case to one of general paralysis was obvious during life, but it was not a typical case, and notwithstanding the absence of syphilitic history it was suspected to be syphilitic and treated accordingly. The necropsy therefore was looked to with interest. There were no external marks of disease. The calvaria was unsymmetrical owing to considerable thickening of the bone overlying the right fissure of Rolando; this thicken-



ing caused not only external prominence, but also encroached on the cavity. The dura mater and sinuses were normal. There was considerable excess of subarachnoid fluid over so much of the convexity of both hemispheres as corresponded to the following convolutions, which were manifestly wasted: the posterior half of the 1st frontal, the posterior end of the 2nd frontal, the upper two-thirds of the descending frontal, and the upper third of the ascending parietal. There was also considerable jelly-like œdema at the base of the brain, embedding the basilar artery, the circle of Willis, and the optic chiasma. There was no recent meningitis, but the opposed surfaces of the frontal lobes were strongly adherent. The substance of the brain generally was healthy in colour and consistency, but in the anterior end of the right optic thalamus there was a small recent hæmorrhage with petechial spots around, the whole measuring about  $\frac{1}{2}$  inch across, and a similar patch, the size of a pea, was found just under the grey cortex near the hinder extremity of the right fissure of Sylvius. The basilar artery was diseased from end to end, the walls much thickened, white, fibrous, but not calcified, and it was lined throughout with an adherent layer of old decolorised clot, which peeled off like a diphtheritic membrane. The first inch of each middle cerebral was similarly diseased, but contained no *antemortem* clot. The spinal cord and membranes were healthy. Beyond slight atheroma of the first part of the aorta and congestion of the base of both lungs no further disease was discovered.

The second case was that of a married woman, aged 46, who came under my care on February 24th, 1887. There was no history of antecedent illness. About two years previously she began to suffer from headache, and some change in her mental condition was observed. Twelve months later she consulted Mr. Bell-Irving for shooting pains in the back of the head and in her arms. A fortnight after this she fell down in a fit, and remained semi-comatose for the next four days. After this she presented considerable physical weakness, with exaggeration of tendon reflexes and cloni in both lower extremities and tremulous movements in the right arm and leg, and she became forgetful and emotional. About seven weeks before admission she had a second fit, attended, like the former, with unconsciousness lasting for several days. After this fit her mental powers still further



deteriorated, the relative weakness of the right arm and leg increased, she suffered from attacks of headache followed by shivering, and her eyesight, which had been failing for a year or two, became much impaired. Her temperature while under Mr. Bell-Irving's care never rose above the normal. On admission, the patient was a thin woman, unable to give any account of herself, answering slowly and with hesitation, and soon stopping, either from confusing words or from uncertainty of memory. There were no obvious tremors of the lips. There was no facial paralysis, but the head was turned to the right, and there was conjugate deviation of the eyes in the same direction. Rotatory nystagmus was sometimes observed on both sides. The right pupil was larger than the left, but both pupils acted to light. She appeared to see obscurely, but could not count fingers. Both optic discs were atrophied; there was no definite paralysis of the limbs, but the grasp of the right hand was weaker than that of the left, and in standing or walking (which she would only attempt when strongly supported) she had a tendency to lean or fall to the right. No anæsthesia; knee jerks brisk; ankle clonus on right side; pulse 104, small, tense; urine, specific gravity 1012, with a trace of albumen and a few hyaline casts. In the evening she had a fit, in which she was unconscious for a few minutes and was slightly convulsed. The temperature rose to 100·2°. During the next fortnight there was little change in her condition; she was generally drowsy, but at times fully awake, and although she was forgetful, weak-minded, did not know where she was, and suffered from delusions, would at these times generally answer questions readily and even flippantly, showing some sense of humour and readiness of retort. Occasionally she would get out of bed and grope her way round it, and was apt whilst in bed to pick at things, especially with the right hand, and to sit up and turn round, mainly to the right, and to grope and fidget with her hands as though she were engaged in disentangling herself from things about her. The nystagmus and deviation of head and eyes continued, but on one or two occasions the right eye was seen to turn in to the inner canthus. The left never even reached the middle line. The pupils still acted to light, but she had apparently become quite blind. The right hand still trembled when she tried to use it. When she stood she still had a tendency to fall to the right. She took food well, but never tried to help herself. She

appeared to have power over the rectum and bladder if she cared to exercise it, but for the most part she passed her evacuations incontinently. She had no headache or sickness, and her urine still presented a trace of albumen. Between March 12th and 24th she became fidgety and talkative, and at the same time the right arm became more tremulous than it had been, and the fingers clenched, and the grasp of the left hand became markedly feeble. During the next two or three days the paralysis of the left side gradually increased, the right arm at the same time becoming more rigid. It was noted too that the left pupil was larger than the right and did not act to light, and that the right eye still occasionally moved independently to the inner canthus. She was sick at times and gradually grew more and more drowsy and stupid. On October 24th the eyes were examined again. At this time the right pupil was the larger, both pupils acted to light, and the discs presented as before much grey haze at the margins, which could not be defined. On the 26th the muscles of the face quivered a good deal. On the 28th she was absolutely unconscious and lay with her mouth open, and was unable to swallow solid food. She was now evidently sinking; her pulse became rapid and weak; her respirations assumed the Cheyne-Stokes character; her eyes twitched and still deviated to the right and presented rotatory nystagmus; her tongue within her mouth was constantly jerking forwards and to the left at the rate of thirty-four in the minute; her temperature, which had been normal, rose to  $103.8^{\circ}$  shortly before death, but had fallen to  $101.4^{\circ}$  at the time of death, which occurred on the morning of March 29th.

*Necropsy.*—Brain: The skull, dura mater, and venous sinuses were all healthy. The arteries were very atheromatous, and here and there were much narrowed. This was especially the case with both middle and both posterior cerebrals, certain parts of which were white and cord-like. The membranes about the optic nerves were opaque, but otherwise healthy. The brain generally appeared somewhat shrunken. Right hemisphere: The middle third of the ascending frontal convolution was softened to a depth of  $\frac{1}{3}$  inch. There was extensive softening of the inner aspect of the occipital lobe, reaching as far forwards as the internal perpendicular fissure and involving in its whole thickness the cerebral substance lying between it and the posterior cornu of the lateral ventricle. A patch of discoloration without softening was found



in the optic thalamus. Left hemisphere : An area of softening— $2\frac{1}{2}$  inches vertically by 1 inch horizontally—involved the occipital lobe just behind the angular gyrus and the lower half of the superior parietal lobule and extended deeply. There were two patches of softening in the ascending parietal convolution, one  $\frac{1}{2}$  inch above the fissure of Sylvius, the other adjoining the softened area in the superior parietal lobule. They were each about  $\frac{1}{2}$  inch in diameter. There was superficial softening of the upper part of the cerebellum to the left of the median line and of the lateral points of the lateral lobes. The softened tissues had entirely lost their normal characters, presenting yellowish discoloration in patches, and here and there small hæmorrhages. All other parts of the brain were healthy. The heart weighed 16 ounces, its left ventricle was hypertrophied, and there was a little thickening of both aortic and mitral valves. The kidneys were granular and weighed together  $7\frac{1}{2}$  ounces. No other disease was found.

I admit that the opinion I have ventured to form as to the etiology of these latter cases may not commend itself to everyone, and that, as to the last especially, it may reasonably be held that the arterial disease was simply related to the contracted granular kidneys. But, on the other hand, the disease in both cases was limited strictly to the arteries of the brain, and in its character and distribution was practically indistinguishable from such as one meets with in syphilis. I must admit also that no history of syphilis was elicited in either case, but then some of the most typical and striking examples of tertiary syphilis that have come under my observation have been equally defective in the matter of history. I may further remark of the first case, that its duration of only eight months was very short for one of general paralysis of the insane. And I may here confess that as regards the second case my diagnosis as to the seat of disease was wholly at fault. Misled by the association of relative weakness of the right arm and leg with conjugate deviation of the eyes to the same side, I assumed that the patient had some softening or tumour in the pons; and when, finally, more definite paralysis struck the other side I was only confirmed in my error, assuming as I did that the disease in this part had spread laterally. It is not difficult, however, to understand how extensive lesions of the surface of the brain and lesions limited to the pons may be attended with symptoms of striking similarity. I have not taken the trouble to



tabulate all my cases of syphilitic disease of cerebral arteries; but the few which I have quoted confirm the views of those who hold that such disease may come on at any time after the generalisation of the syphilitic virus, in cases of inherited syphilis as well as in those in which the disease has been acquired, and independently of age or sex influence.

There are one or two points of interest presented by the cases I have adduced which seem to me to be deserving of consideration. It is, I believe, generally held that when one of the cerebral arteries becomes obstructed the district supplied by it undergoes softening and may become the seat of hæmorrhage, and that the district thus affected, unless it be a very small one, never becomes reinstated. It is obvious, however, that this is a rule which is liable to exception; for in the remarkable case which I narrated in my last lecture, in which the intra-cranial portions of both internal carotids and their branches and the basilar were all obstructed by old clots, which, both from the history and *post-mortem* evidence, must have formed at different periods, there was no trace of softening or of any other pathological change in the nervous centres. I may add that cases are occasionally met with in which patients do recover perfectly from the effects of embolic obstruction of one of the cerebral arteries. It is an interesting fact also that whilst softening of brain-substance or hæmorrhage was usually observed in distinct association with the obstruction of an arterial trunk or branch, similar lesions were by no means unfrequently present in regions the arterial service of which appeared to be sound. Thus, in Dr. Hawkins's case, there was some disease in both internal carotids, the other arteries being healthy, but there was hæmorrhage into both lateral lobes of the cerebellum. In my fifth case there were old obstruction of the left internal carotid and a cyst in the left corpus striatum, but there was also softening of the right corpus striatum, the arteries leading to which were healthy. In my sixth case the right middle cerebral was obstructed, and there was softening of the middle lobe of the corresponding cerebral hemisphere, but at the same time a cyst in the left corpus striatum. In my eighth case the posterior cerebral was thickened and reduced in calibre, and there was softening of the corresponding temporo-sphenoidal lobe, but there was softening also in the pons; and, lastly, in the second case, which I have cited to-day, there was, no doubt, the association

of widespread softening of cerebral and cerebellar tissue, associated with widespread disease of arteries, yet none of the vessels were found to be actually obstructed, and softening was present in regions the arteries of which appeared to be wholly unaffected. The explanation I am disposed to offer of the phenomenon is the obvious one that the nutritive lesions were determined less by disease of the larger vessels than by disease of the smaller vessels leading to and ramifying in the affected districts, and that it is these latter which are often alone affected in particular districts. I base this view partly on the fact that such disease of the smaller vessels has been observed, partly on the difficulty there is in otherwise explaining the phenomenon, and partly on the consideration that it serves to link together cases which both on clinical and on anatomical grounds seem at first sight to have little connection with one another. I may also add that in Dr. Hawkins's case and in my case of disease of the coronary arteries of the heart, there were infarcts in the lungs and in the kidneys. Now in neither case was there pyæmia or source of embolism. But in Dr. Hawkins's case there was blocking up of the renal arteries and disease of the ramifications of the pulmonary artery, to the presence of which the infarcts were doubtless attributable. In my case no such disease of pulmonary or renal arteries was discovered, but it seems to me reasonable to assume that the hæmorrhage was determined by obstruction of the smaller or microscopic arteries. I have seen infarcts in the lungs and kidneys in other syphilitic cases. In connection with this subject, I venture to recall a case which has been a puzzle to me ever since I made the *post-mortem* examination on it in 1852. It was that of a man aged 37, who was admitted with obscure febrile symptoms, and who died ultimately of pyæmia arising out of an intercurrent attack of cellulitis. There had, I believe, been no symptoms suggestive of brain disease. At the necropsy the brain and its appendages were found healthy, with two exceptions: the one being the presence of undue thickening and opacity of the sub-arachnoid tissue, the other that the minute arteries in the cerebellum and corpora striata were almost universally calcified and projected from the cut or broken surfaces like needles. Beyond the evidences of pyæmia there were no traces of disease throughout the body. I have often wondered whether this condition, unique in my experience, could have been a consequence of



old syphilitic disease of vessels. I by no means venture to commit myself to an opinion. I simply put a curious case on record and make a suggestion.

This short discussion on the subject of syphilitic disease of the capillary arteries leads me to make a remark or two on syphilitic disease of the veins. I am not aware that any special attention has been given to this affection, and I confess that I have very little to say about it. Mr. Hutchinson, in his book on 'Syphilis,' gives a short account of "periphlebitis as a consequence of syphilis," and, without quoting his description, I may say briefly that I have notes of a case or two which probably belong to the same category; especially that of a gentleman between 40 and 50 years of age who had a chancre twenty-five years previously, who had had what he termed "boils" on his legs, leaving deep scars, some ten or fifteen years later, and, apparently originating about the same time, large varicose veins in one leg below the knee. He consulted me for a very serious syphilitic outbreak, limited to that leg, in which unquestionably the veins were implicated. I recollect also a very interesting case which I published some years ago of a man of middle age who came to me with symptoms suggestive of an intra-thoracic tumour. He had obstruction of the superior cava or both innominates, with great dilatation of the veins of the neck and of those in the thoracic walls, much congestion and œdema of the head and neck, and severe dyspnœa. But he had had syphilis, as was shown by ulcerative destruction of the palate, and he was cured speedily by anti-syphilitic treatment. I assume that in this case there were gummata about the base of the heart obstructing the veins either by pressure or by extension of disease into their walls. I may further point out that the cerebral venous sinuses are often similarly involved in the progress of gummatous affection of the dura mater. I cannot call to mind any case in which such obstruction has of itself led to any serious consequences; but I shall presently narrate a case in which the cavernous sinus appeared to have been obliterated. Dr. Hawkins's case of apparently syphilitic obstruction of the renal arteries has brought to my recollection a case of thrombotic obstruction of the renal veins which may have been syphilitic (but of that you will be able to judge as well as myself), and is certainly worth recording. The case was that of a woman, aged 37, on whom I made a *post-mortem* examination in March, 1852. She had



tubercles in her lungs, but none anywhere else. The kidneys were much enlarged, measuring about  $5\frac{1}{2}$  inches in length,  $3\frac{1}{2}$  inches in breadth, and 2 inches in thickness. Their surfaces presented several irregular depressed blackish patches. On section the cortical substance was pale, succulent, and in many places reticulated, as though the tissue had become rarefied and the interstices filled with serum. "They presented several opaque whitish circumscribed patches, which had the appearance of tubercular, or perhaps more properly of fibrinous, deposits, being firmer and less cheese-like than the former," and they corresponded to the depressions seen on the surface. The renal veins were much thickened and in their whole extent filled with old decolorised adherent clot. On tracing the branches into the kidney these, almost to their smallest ramifications visible to the naked eye, were similarly filled. The clots were prolonged from the veins into the cava, where they presented rounded ends which were turned upwards in the direction of the blood-stream and came into contact with one another and partly blended. I gave a microscopic description of the parts, which it is not worth while to refer to here.

In the above account I have quoted my original words. I have no doubt now, as I evidently thought then, that the patches in the kidneys were not tubercular. Were they gummata or simply old infarcts? Had the obstruction been in the arteries there could be little difficulty in deciding in favour of the latter alternative. But venous obstruction does not, so far as I know, cause infarcts. The patches were just like gummata in the spleen or liver; and when I used the expression "fibrinous deposits" I was undoubtedly thinking of what were then termed "knotty tumours" of the liver—tumours which we now know to be syphilitic.

I do not propose to describe gummata. It is sufficient for my purpose to remark that they develop mainly in connection with the dura mater or other membranes of the brain, and tend, according to their seat of origin and direction of spread, to become embedded in the brain substance, to grow along and involve nerves, and to implicate the venous sinuses and even the arteries; that they form tumours varying, roughly speaking, from the size of a pigeon's egg to that of a pin's head; and that in their origin and progress they are apt to be associated with more or less of what resembles ordinary inflammatory process and exudation which

extend beyond and around them, causing thickening, adhesion, and matting together of membranes, and infiltration of neighbouring nervous tissue. It might be difficult to determine in any case how far these latter conditions are truly specific and how far they are due to simple inflammation excited by the irritation of the gummatus growths. But considering that these are essentially only inflammatory growths due to the irritation caused by the specific virus, and that their characteristic microscopic and coarser features are by no means so universally distinctive as to render their diagnosis in all stages of their development and in all situations a matter of certainty, it may reasonably be assumed that these outlying and sometimes independent tracts of apparently simple inflammation are in large proportion, if not always, themselves specific.

My first acquaintance with cerebral gummata was made in March, 1854, when I conducted a *post-mortem* examination on a patient under the care of the late Dr. T. A. Barker. The case was one of cerebral disease, but no clinical record has been preserved, and I need scarcely say that neither I nor anyone else recognised the true significance of the lesions which I found and described. These, however, clearly interested me very much at the time and I described them carefully, and my description is conclusive as to their nature. The patient was a woman aged 30. There was no disease of the thoracic or abdominal viscera. The calvaria was healthy, and so also were the membranes and surface of the brain seen *in situ*. On removing the brain, however, it was observed that several of the nerves at the base were diseased and also that the basal dura mater was affected in patches. The right 3rd nerve, from its apparent origin to a short distance within the cavernous sinus, was irregularly enlarged to three or four times its normal diameter, somewhat indurated, of a yellowish-grey tinge, markedly translucent, and slightly vascular. The left 3rd was affected similarly, but to a less extent. The right 5th nerve was for the most part natural, but its trunk presented a small fasciculus of diseased fibres. The left 5th, with the exception of its motor part, was affected in its whole thickness for about  $\frac{1}{2}$  inch of its length, beginning from its apparent origin. The diseased part was indurated, thickened and irregular. The distal part of the nerve was normal in character but reduced in size. A few of the branches of origin of the right pneumogastric were affected, and the left spinal accessory was very considerably



diseased. All the other nerves were healthy. There were patches of diseased dura mater on the hinder surface of the posterior clinoid processes, on the under aspect of the tentorium cerebelli at its attachment to the apex of the right petrous bone, and on each side of the foramen magnum. These appeared to have originated in the free surface of the membrane, were severally about the size of a silver penny in area, but irregular in outline, a line or two thick, and nodulated on the surface. They had a translucent jelly-like appearance, a yellowish-grey tint, and were very soft. A few small nodules of like character studded the visceral arachnoid about the pons and at the commencement of the vertebral canal; and one the size of a pea sprang from the left vertebral artery. It appeared to me that the disease was everywhere of the same character, and that the morbid material infiltrating the diseased nerves was identical with that forming independent outgrowths. I described them as consisting microscopically of an interlacement of delicate fibres, thickly studded with round or oval nuclei about as large as blood-corpuscles. The brain substance was universally healthy. The arteries at the base presented a few spots of atheroma.

In February, 1859, I examined a case under the care of the late Dr. Peacock which may be quoted here, although I do not pretend to know what the nature of the disease was. The patient was a single woman aged 23 who no doubt died with symptoms of cerebral disease, since I was allowed only to examine the brain; but no clinical account of the case has been preserved. The dura mater was adherent at many points to the brain, and on removing it many fragments of brain substance (corresponding to these points) were removed with it. The surface of the brain was studded with pearly, cartilage-like bodies, rounded or lobulated in form and varying in size between that of a poppy-seed and that of a tare. They appeared to be connected mainly with the pia mater. They were sparsely scattered over the general surface of the cerebrum, but were more abundant over that of the cerebellum. They were most numerous at the base, especially about the olfactory nerves, along the Sylvian fissures, in the region of the circle of Willis, and on the surface of the pons and medulla oblongata. They had a tendency to be connected with the vessels and to beset the nerves; thus, they encased the optic nerves, studded the 5th pair very thickly and (though in a somewhat less degree) all the other nerves. A few were present in the choroid plexuses, and



where the brain adhered to the dura mater there were growths of the same nature in the dura mater itself. The brain generally and the arteries at the base were healthy. I described the bodies as consisting of blood vessels, nuclei and fibroid tissue, the nuclei being for the most part globular, refractive and nearly homogeneous. I have never seen a similar case. I was quite sure and am still sure that it was not a case of tubercle. But I am not equally sure that it might not have been syphilitic.

The first case with a clinical history which I shall bring before you is that of a man aged 28, who came under my care on March 19th, 1890. He was not in a condition to give a trustworthy account of himself, and the following history was obtained partly from himself but mainly from his friends. He had had syphilis, but when and whether he had had secondary symptoms could not be ascertained. In December, 1888, a hard lump formed on the top of his head, which after a time subsided without ulceration, leaving a deep depression. In June, 1889, he had a fit, and for several days afterwards complained of weakness and of pain in the left temporal and frontal regions. In July he had a second fit, beginning with a cry and attended with left-sided convulsions. A third fit occurred a month or two later. During all this time, excepting while suffering from the immediate effects of his fits, and till Christmas, 1889, he continued at work. At this date the headache, from which he had continued to suffer off and on, became much more severe and persistent and his spirits depressed. In the beginning of March, 1890, he became strange in his talk and behaviour. On admission he was a thin, pale man, lying quietly in bed in a drowsy state, answering questions slowly and incoherently and complaining of headache. His articulation was slow and hesitating, and he spluttered over some of his words, so that at times it was impossible to understand him; but there were no tremors of the lips or tongue. The right pupil was larger than the left, and neither acted to light nor, so far as could be made out, to accommodation; there was no paralysis of the eye muscles, nystagmus, or abnormality of the discs, but there was a small patch of choroiditic atrophy in the left yellow spot region. A depression  $\frac{1}{3}$  inch deep and admitting the tip of the finger was found near the centre of the vertex, but was free from tenderness or scar. There was no trace of paralysis or of affection of the nerves or of scars or eruptions on the skin. The abdominal and

thoracic organs appeared healthy, and he passed his evacuations consciously. No change occurred until April 11th, when it was observed that there was weakness of both internal recti. On the 15th I noted "that he had been getting weaker and more drowsy and stupid and subject to delusions; that he had distinct paralysis of both internal recti, but mainly of the right one, the right eye being incapable of moving inwards beyond the middle line; that there seemed also to be slight impairment of the upward and downward movements and a tendency for the right lid to droop." On the 18th, for the first time, the lower part of the right side of the face, the right arm, and right leg were manifestly weaker than the corresponding parts on the left side, but the tongue did not deviate. He was now beginning to pass his evacuations into the bed. He continued gradually to get worse, more and more apathetic and drowsy and difficult to rouse, though still presenting occasional intervals of comparative brightness and intelligence; the paralysis of his 3rd nerves increased, especially in respect of the inward and upward movements of the eyes and the raising of the eye-lids, although even to the last he could elevate the latter with an effort; the right-sided hemiplegia remained without change. On May 5th, on the evening of which day he died, he was in a state of complete coma, lying on his back with eyes divergent and incompletely closed, breathing quickly, with rapid pulse, perspiring skin, and an occasional slight cough. His temperature, which had generally been subnormal, rose a little during the last few days of life, and shortly after death (which took place at 10.30 P.M.) was found to be 107°.

*Necropsy.*—There was no cutaneous scar at the seat of the depression in the vertex; but the subcutaneous tissues, including the periosteum, were thickened and scar-like. The brain generally, and its membranes and vessels were perfectly healthy; but at the base there were several gummata, apparently originating in the pia mater, though more or less embedded in the adjoining nerve or brain tissue. They were all caseating internally, but presented thin translucent pinkish vascular walls. One was situated close to the right optic foramen, and was embedded, on the one hand, in the cortex at the junction of the tip of the temporo-sphenoidal with the frontal lobe, and, on the other, involved the optic nerve from its outer side to its centre, the nerve here being swollen to twice its normal size, and adherent to the edges



of the foramen. Another surrounded, but did not obliterate, the left Sylvian artery, at about the spot at which the branches are given off to the central ganglia. A third was situated just beyond the hooked extremity of the left hippocampal convolution. A fourth, situated near the posterior extremity of the left occipital lobe, was embedded in the cortex and also adherent to the dura mater. The fifth involved both corpora albicantia, and sinking deeply involved also the junction of the crura cerebri as they emerged from the pons. The 3rd nerves seemed to originate in or close to it. The tumour was fairly symmetrical, but rather larger on the right side than on the left. Beyond the facts that the pleuræ were adherent, that there was hypostatic congestion of the bases of the lungs, and that the liver was adherent, superficially puckered, and presented strands of cicatricial tissue in its substance, nothing noteworthy was observed elsewhere in the body.

My next case is one that excited a good deal of interest during life. It is that of a married woman, A. M——, aged 49, who came under my care on July 22nd, 1884. She had had a large family of children, of whom four had died as infants; but no history of syphilis could be elicited. She had suffered, however, from deafness in both ears for about seven years, and she had an eruption on the back of one of her hands, attributed to an injury a few years previously, but which was certainly syphilitic. Her illness dated from the previous March, when she first complained of some difficulty in swallowing. Early in June she became an out-patient of Dr. Semon's, who found that she had paralysis of the abductors of the left vocal cord; the voice, however, was unimpaired. Dr. Semon was at this time naturally inclined to attribute her laryngeal affection to some intra-thoracic lesion. At the beginning of July she found she could not open her left eye, and then placed herself under Mr. Nettleship's care. Down to this time the symptoms had been limited to the domains of the left recurrent laryngeal and 3rd nerves; but now they began to make rapid progress, and when admitted three weeks later the following was her condition:—She was much emaciated, and there was complete ptosis of the left upper eyelid and an internal strabismus of the corresponding eye. This could be moved very slightly upwards, downwards, and inwards, but not at all outwards. There were nebulæ in the cornea. The right eye was healthy in every respect. The sight of



both eyes was fairly good, and the pupils were equal and acted to light and accommodation. There was marked impairment, but not absolute loss, of feeling over the area of distribution of the left 5th nerve, and the temporal and masseter muscles of this side contracted feebly, and the chin (when the mouth was opened widely) was thrown over towards the left. There was impairment of smell in the left nostril, and of taste in the left half of the tongue. This organ was protruded straight. The voice was reduced to a hoarse whisper, and the left cord was paralysed, mainly as regards its abductors. She had some difficulty in swallowing. The deafness in both ears was considerable, but equal. There was no paralytic affection of the limbs or trunk, and, though complaining of giddiness, she walked without difficulty. She had much pain at the vertex and on the left side of the face. There was no further sign of disease, and her digestive and other functions were all in excellent condition. During her stay in the hospital repeated examinations were made, and the following additions or corrections as to her symptoms were noted:—She had paralysis of the left half of the soft palate, almost complete anæsthesia of the left half of the tongue, mouth, and fauces, and impairment of the sensibility of the epiglottis and larynx mainly on the left side. The facial pain, which was referred to the left ear and eye and to the lower jaw, was evidently due to implication of the 5th nerve. It was only after she had been in the hospital for three weeks that attention was called to the eruption on the right hand. But from the beginning it had been suspected that the case was syphilitic, and she had been treated accordingly. She improved in health, and lost her pain and difficulty in swallowing; but no further improvement had taken place when she left the hospital on September 27th. She went on fairly well until the beginning of November, when she had three fits attended with loss of consciousness, after which she became very weak and ill and unable to walk. On December 9th she had another fit, and was readmitted. She was now sallow, emaciated, exceedingly weak, and unable to stand; but the limbs were not paralysed. In other respects there was very little change. The anæsthesia of the left side of the face, tongue, fauces, and inside of the mouth was complete. The paralysis of the left half of the soft palate was more pronounced than it had been. The tongue was still protruded straight. Her voice was inaudible, but she swallowed

without difficulty. Dr. Semon reported of the larynx as follows :—“The left vocal cord stands near, but not quite in, the middle line. On attempted phonation the right moves slowly towards the middle line, but does not cross it, and there remains a gap between the inner borders of the cords.” The paralysis of the left temporal, masseter, levator palpebræ, external rectus, and superior oblique still continued, and Mr. Nettleship noted that the superior, inferior, and internal recti still acted a little. She had no recurrence of fits, and little, if any, pain; she was never sick, but had absolute loss of appetite; she got more and more apathetic, and weaker and weaker, until at last she died of exhaustion on January 31st.

*Necropsy.*—On removing the brain extensive disease was found at the base of the skull. The dura mater in the middle fossæ, sella turcica, and over the basilar process was much thickened, in places to at least  $\frac{1}{4}$  inch. The thickened tissue was for the most part firm and semi-translucent, but here and there softer and pinkish in hue. The thickening was especially pronounced in the course of the left cavernous sinus, and the 3rd, 4th, 5th, and 6th nerves were embedded in it, and were followed and recognised with great difficulty. The corresponding nerves on the opposite side were also embedded in thickened membrane, but could be readily traced, and appeared healthy. There was considerable thickening about the foramina ovalia. The vagus, glosso-pharyngeal, and spinal accessory at its junction with the vagus on the left side were embedded in firm fibrous material, as also (but in less degree) were their fellows on the right side. The olfactory, ophthalmic, facial, and auditory nerves were all free. On removing the thickened dura mater, especially on the left side, the bone was found eroded and as if worm-eaten. The fibrous tissue around the left cavernous sinus and in connection with the sella turcica was extremely thick, and contained here and there in its substance a yellow glairy fluid, looking a little like pus, but presenting only fatty detritus. The sinus itself could not be traced. The nerves in the orbits appeared healthy. The pia mater, vessels, and substance of the brain and cord were healthy. In the outer and lower part of the upper lobe of the right lung was a cavity about an inch in diameter, well-defined, thick-walled, and full of dirty-yellow fluid; and the surface of the lung corresponding to it was loosely adherent to the chest walls. It was thought to repre-



sent a softened gumma. There were no tubercles. The capsule of the liver was thickened, and the kidneys were slightly granular. The left aryteno-epiglottidean fold was somewhat thickened. The left crico-arytenoideus posticus and crico-arytenoideus lateralis were atrophied and of a yellowish tint. The recurrent laryngeal and vagus looked healthy. All other organs were healthy.

This case is interesting from several points of view: one being the fact that the very first indication of intra-cranial disease was paralysis of the left recurrent laryngeal, leading to the suspicion that she was suffering from intra-thoracic disease; another being the fact that paralysis of the left side of the soft palate was associated with paralysis of the motor nerve of the larynx on the same side, and was wholly independent of lingual or facial paralysis, implying, what has of late years been proved experimentally, that the motor nerves of the soft palate and of the intrinsic muscles of the larynx are derived from the same source; a third being the fact that the cavernous sinus was involved in the syphilitic lesion and occluded.

The last two cases were cases of fairly rapid progress, and much of their interest depended, less on the actual amount of disease present within the skull than on its situation and the implication of particular nerves. In my next case the intra-cranial disease was much more chronic; the amount present was very considerable, and especially there was a very large tumour developed, apparently, within the substance of the brain. The case was my own, but was recorded by Dr. Greenfield in vol. xxviii of the 'Transactions of the Pathological Society.'

A. G.—, a sawyer, aged 34, came under my care on October 5th, 1875. He had had double otorrhœa after scarlet fever when a child, and had remained liable to it since. He had had a chancre but no rash. He had, however, had soreness of the throat and palate. Four years ago he was attacked with severe pain in the vertex, occiput, and neighbourhood of the left mastoid process; he became deaf in the left ear; and on waking one morning he found that he had lost the use of the left side of the face. These symptoms continued with little variation till the time of admission. Latterly, however, his sight had become defective and he had acted strangely. On admission there were marks of ulceration on the throat and palate, and hard glands in both groins. There was paralysis of the left portio dura, and the uvula pointed to the right. No other evidence



of paralysis was obtained. His sight, however, was imperfect, he had double optic neuritis, and he was deaf of the left ear. He was sensible, and complained of constant severe headache and of much tenderness in the left mastoid region and over the vertex and the spines of the upper cervical vertebræ. As time went on, his blindness increased, paralysis of the left half of the soft palate became unmistakable, and the left external rectus became paralysed. Later it was ascertained that vision was specially impaired in the upper half of each retina, his left pupil became dilated and motionless, and his left arm and leg partially paralysed. During his residence in the hospital his headache in the situations above mentioned and the pain in the back of the neck continued, and he was restless and often violent. He died on December 20th.

*Necropsy.*—The calvaria was thick, very dense, and firmly adherent to the dura mater, on removal of which its exposed surface was found rough and irregularly pitted. The inner aspect of the dura mater, especially over the right hemisphere, was adherent to the brain by a thin soft layer of lymph, and was studded here and there, but mainly in the course of the sinuses, with irregular nodulated outgrowths, of which some were embedded in the convolutions. These were yellowish, and obviously gummata. The channel of the left lateral sinus was much narrowed by the presence of such growths in its walls. The cerebral convolutions were somewhat flattened, the pia mater injected, and there was excess of subarachnoid fluid. In the posterior extremity of the right hemisphere, occupying the greater part of the occipital lobe, was a rounded gummatous tumour, about  $1\frac{1}{2}$  inches in diameter. It was for the most part firm, opaque, and yellow, but here and there was greyish-red, translucent, and soft. It nowhere quite reached the surface. The pia mater over the right hemisphere in irregular patches was infiltrated with a translucent yellowish material; and on close examination it was seen that many of the vessels connected therewith were thickened and converted into white cords. Excepting that there was softening in the neighbourhood of the embedded gummata, the brain was generally healthy, as also were the arteries at the base of the brain. The optic nerves were somewhat swollen and the left tract wasted and softened. The right 3rd nerve, where it passed round the crus, was slightly reddened, swollen, and soft. Both 6th nerves were flattened in their course at the base of the skull, especially the left, which was atrophied. The

other nerves appeared healthy. There was no disease of the thoracic or abdominal organs. Presumably the left facial and auditory nerves were involved in the thickening of the dura mater, which also involved the left lateral sinus.

It is curious that there is no record of the occurrence of fits, which are so common and almost characteristic of the presence of gummata. It is curious, too, that until quite a late period the only paralysis present was that of the left facial nerve.

In connection with the subject of large cerebral gummata, of which the last case furnishes a noteworthy example, I will make a brief reference to another case that came under my care in 1880. The patient was a young woman who was transferred from King's College Hospital to St. Thomas's Hospital on July 14th. I received no history with her beyond the statement that she had been suffering while in King's College Hospital from unilateral epileptiform attacks. On admission she was semi-comatose. She did not speak or take notice excepting when attempts were made to move her limbs or open her eyelids, which she resented. She had no definite paralysis, but swallowed with difficulty. The right pupil was larger than the left, and neither acted to light. She had double optic neuritis. Her temperature on admission was  $99.6^{\circ}$ ; the next morning it was  $101.6^{\circ}$ . Towards the afternoon of this day she sank into profound coma and her temperature rose rapidly; and just before death, which took place in the evening, it reached  $107.4^{\circ}$ . On *post-mortem* examination depressed scars were noted on the forehead, chest, and back, and there was a syphilitic gumma as large as a hen's egg embedded in the right anterior cerebral lobe, with thickening and adhesion of the membranes over it. The vessels were healthy. The only other evidence of disease was the presence of scars on the surface of the liver and spleen.

Before I conclude my lecture, I should like to quote one other case, in this instance not fatal, which presumably was much of the same character as the last but one; for in its course the ears became affected, both facial nerves became paralysed, she had double optic neuritis and hemiplegia; but additionally she had marked evidence of syphilitic involvement of the periosteum of the skull.

A married woman, aged 25, was admitted under my care on March 19th, 1878. After the birth of her first child she had an eruption on the legs and her hair came off. The child died when



seven months old and was said to have been syphilitic. Six months previously she had a fall from a trap, struck her head and face, and was stunned and insensible for two hours; but she recovered completely in a short time. On January 1st she awoke in the morning with violent pain at the top of the head, where also it was swollen and tender. An incision was made, but only a little blood escaped. Since then the pain has been almost constant. She was healthy looking, but complained of severe pains in the forehead and sides of the head and face and in the eyes, ears, and teeth, which were worse at night, and of which the pains in the eyes and ears were aggravated respectively by light and noise. Her eyelids were swollen and there was puffiness and considerable tenderness over the forehead and sides of the head. She was quite sensible. There was no paralysis or anæsthesia. She had double optic neuritis. On April 27th it was remarked that the severe pain continued in the head, ears, and teeth; that the headache was apt to shift, being worst sometimes in front, sometimes behind, sometimes at the sides; that there was much tenderness and œdema, especially about the zygomatic regions; that the temporal muscles acted imperfectly; that a tender swelling had appeared towards the back of the left side of the head; and that she was frequently sick. On May 26th a note was made to the effect that the pains had continued and were sometimes so severe (especially at night) as to make her scream, at which times, also, she was apt to be delirious; that during the last few days the pain had been localised mainly in the mastoid regions; and that her sight was failing. About a week later it was observed that she had commencing paralysis of the left portio dura, and that she complained of diplopia. At this time it was also noted that the discs were hazy, their outlines blurred and surrounded by a grey zone; that there was some swelling; and that the vessels were tortuous and the veins enlarged. The facial paralysis increased during the next week or two and then slowly subsided. In the beginning of July (by which time the œdema and tenderness had disappeared from the frontal, temporal, and zygomatic regions, and there was scarcely a trace of left facial palsy) she complained of pain and stiffness in the back of the head and neck and of severe pain in the ears. Soon after this, the pains continuing, she became deaf on both sides; on July 18th it was observed that since the day before she had been completely deaf and had had noises in her ears;



and on or about the 25th she had copious discharge from both ears, and she could not quite close the right eye. From this date improvement, on the whole, continued; she soon lost all pain in or about the head, and she expressed herself as feeling quite well but for the gathering in her ears and "the beastly taste in her mouth." But the discharge from the ears continued, both membranæ tympani were found to be perforated, she remained almost absolutely deaf, and the paralysis of the right portio dura became almost complete. She left at her own wish on August 3rd, having been in the hospital nearly five months. The patient again came under my care six years later. She then stated that but for persistent deafness and occasional discharge from her ears she had quite recovered and had continued well until recently, when she had recurrence of severe headache. This became aggravated a fortnight before admission, and was attended with return of discharge from the left ear. A week later she was seized with vomiting, and observed that her right arm and leg were weak. On admission she was restless and moaning with pain, which she referred to the forehead and left side of the head, suffered from vomiting, and presented complete left facial palsy with slight but obvious weakness in the right arm and leg. There were traces of the old optic neuritis. She again improved under treatment, and left well but for persistent facial palsy.

In this case there was evidently abundant syphilitic affection of the periosteum of the skull, and not improbably of the soft parts superficial thereto. The middle ears were also implicated; and there can be little doubt that the dura mater shared in the mischief. The variable facial palsy and the deafness may have been due to the ear affection. On the other hand, they may have resulted from implication of the nerves in disease of the dura mater, an explanation which may equally apply to the pains in the domain of the 5th pair. The optic neuritis and temporary right hemiplegia point, of course, to the presence of intra-cranial disease. I need scarcely say she was treated systematically on both occasions with mercury and iodide of potassium.

---

## LECTURE III.

MR. PRESIDENT AND GENTLEMEN,—In the lectures which I have had the honour of delivering before you I have quoted cases of what may be regarded as typical syphilitic pachymeningitis, notably those of A. G—— and A. M—— included in my last lecture. But in the former of these the meningitis was complicated, as is generally the case, with gummatous outgrowths; and in the latter, although there were no such definite tumours, the dura mater was the seat not of ordinary inflammatory, but of characteristic gummatous, infiltration. I see no sufficient reason, either clinical or pathological, for making any distinction between such cases and those of ordinary gummata of the dura mater; and, in announcing that I would devote the earlier part of the present lecture to the subject of syphilitic pachymeningitis, my main object was to give myself the opportunity of bringing before you a case in which chronic thickening of the dura mater of undoubted syphilitic origin was present, yet free from any of the characteristic traits of that affection. I have previously referred to the facts so often exemplified in the liver that not only are true gummata apt to be surrounded by, and imbedded in, cicatricial tissue undistinguishable from the corresponding results of simple non-specific inflammation, but that also such cicatricial tracts may furnish the only evidence of syphilitic disease; and I have suggested that such apparently simple inflammatory lesions are probably in many cases as truly specific in their origin as gummata themselves.

H. W. G——, a debt collector, 28 years of age, first came under my care on the 16th June, 1887. He had had no serious illness until in January, 1879, he contracted syphilis. In September of the same year he became a patient of Mr. Nettleship's for syphilitic iritis. For this he was under specific treatment in the hospital for many months, and was discharged with little or no adhesion, and otherwise apparently well. In 1883 he was laid up for a time with what was called rheumatic fever, and subsequently was in the hospital for four or five weeks with "sciatica." After this he remained in good health until November, 1886, when one morning he felt a sudden pain in the forehead, lost sight, and fell down. He states that he did not lose consciousness, and that his

sight soon returned, but that he felt very weak, and vomited for several hours afterwards. He was confined to bed from this time to the end of March, suffering from headache which was continuous, though varying in intensity, great muscular weakness, and occasional attacks of nausea and vomiting lasting for a day or two. He had so far recovered by the end of March as to be able to get up, but was unable to do any work. In the middle of April he had another fit, which left him in the same condition as the former one had done. About the middle of May his sight began to fail, and for two or three weeks before admission he was unable to read. He has, however, been gaining strength.

He is a healthy-looking man, suffering from headache mainly across the forehead, aching of the eyes, dimness of vision, and loss of muscular power. There is no definite paralysis, but the grasp of the hands is weak, and he walks slowly and with short steps. But there is no evidence of giddiness or incoordination. The knee jerks are brisk, but no cloni are present. Sensation is everywhere perfect. The bladder and rectum are under control. There is no obvious oculo-motor paralysis, but the patient says he sometimes sees double when he looks to the right. The pupils are equal, and act to light and accommodation. His sight is defective, so that he cannot read, and is not improved by glasses. Intense optic neuritis is present in both eyes. His speech is slow, but not stammering. This, however, he says is natural to him. No tremors of lips or tongue. Hearing good.

During his stay in the hospital he progressively improved; on one occasion only having a slight fit, attended with loss of sight, giddiness, pain in the head and nausea, but without loss of consciousness. He was discharged on the 17th July, the note taken on that day being to the following effect: "There has been great improvement since admission, his headache has been diminishing, his optic neuritis has largely subsided, and he can now read, the exaggeration of the knee jerks has diminished, and he looks and feels fairly well."

He was readmitted on the 1st October, 1889. Excepting that in consequence of an accident he was laid up for a fortnight in December, 1888, he has not been confined to bed since he left the hospital. He has, however, never been quite free from headache, has had fits at long and irregular intervals, and his memory has been defective. Nine days ago the headache (chiefly in the right



frontal region) became very severe, and since then it has continued severe, and he has had frequent fits.

On admission, he was dull and expressionless, but otherwise fairly healthy-looking. He complained of headache in the right temporal region; his memory was bad, and at times he seemed to lose himself, would turn and try to pick up something from the floor, and forget where he was. His sight was very bad, the fields of vision much contracted, and there was abundant evidence of his old optic neuritis. In other respects there was no change, and the abdominal and thoracic organs were apparently all healthy. On the day after admission he had seven fits. These were of short duration. In two or three, which occurred when he was out of bed, he fell down. In those which attacked him in bed he sat up gathering the bedclothes into a bundle in his hands, and twisted round until his face was directed to the back of the bed. He was quite unconscious during the attacks, did not know when they were coming on, and, on coming to, looked dazed and frightened. Although his headache continued in a greater or less degree, and was sometimes severe, he had no return of fits attended with unconsciousness, and, so far as his mental or nervous symptoms were concerned, improved, as he had done when previously under my care. During the first week, however, after admission the temperature rose in the evening generally to  $100^{\circ}$  or  $101^{\circ}$ , but on the 4th it reached  $103.6^{\circ}$ , and on the 5th  $104.6^{\circ}$ , on both of which occasions he complained of feeling cold, and profuse sweating followed. It is probable that these febrile attacks were neurotic. Shortly after admission the fields of vision were carefully examined, and found much contracted, mainly in the right half, suggesting (as Mr. Nettleship observed) disease in the right side of the chiasma.

The rest of the patient's life history has little or no direct relation to his cerebral disease. Nine or ten days after admission he began to complain of cough, and in the course of a few days there was evidence of consolidation of the lower part of the right lung and of pleuritic involvement, and he began to expectorate. It is sufficient to add that a gangrenous cavity formed in the lower part of the right lung, that pyo-pneumothorax became developed, that the patient was tapped on two occasions, the first being on October 30th, when sixty-four ounces of foetid pus were removed, the second on the 4th November, when a portion of rib was re-

sected, and a large but unmeasured quantity of thin, foetid, puriform fluid escaped. He gradually sank, and died on 19th November, remaining sensible to the last.

The membranes at the base of the brain were somewhat opaque, and here and there white streaks, together with adhesions, suggested old, inflammatory disease; but no gross changes were observable. Over the right occipital lobe the dura and pia matres were so firmly adherent to one another and to the brain, that it was difficult to separate them. The cortex involved was softish and yellow, but such changes did not extend into the white matter. The affected area was quadrilateral in shape, limited in front by the angular gyrus, which was not involved, behind by the posterior part of the great longitudinal fissure, below by a horizontal line continued from the Sylvian fissure, and above and in front by the posterior extremity of the superior parietal lobule. No further disease was found in the brain, and the vessels were healthy. The right pleura was obliterated by adhesions, and the wound in the thoracic walls communicated with a pulmonary cavity of gangrenous origin. The left lung was œdematous, the heart and abdominal viscera were all healthy.

It was believed during the patient's illness that he was suffering from intra-cranial syphilitic tumours. And it is possible, of course, that he may have had gummata which had undergone absorption. But, however that may be, there is little reason to doubt that the meningeal thickening and adhesions were of syphilitic origin. The case just narrated leads me to speak of another which I have been inclined to regard as also one of syphilitic pachymeningitis, and in which substantial recovery took place under anti-syphilitic treatment. A labourer, 42 years old, was admitted on the 18th November, 1889. He denied that he had ever had syphilis. About the beginning of September he began to suffer from pain in the spine extending from the middle of the neck to the level of the scapular spines. In the lower part of this region it spread so as to involve both shoulders, and on the right side extended down the arm to the elbow. Early in October the pain ceased in the spine and left shoulder, but became aggravated in the right shoulder and arm, extending now to the tips of the fingers. A little muscular weakness had been coming on for a week or two, but very shortly after the pain had concentrated itself on the right side he found on waking one morning that the arm was almost



completely powerless. The pain subsequently diminished, and he complained of pins and needles along the ulnar border of the hand and little finger, and the radial border of the thumb and index finger. On admission, he was a healthy-looking man, complaining of occasional shooting pains in the right shoulder and down the right arm, with almost complete loss of power in the same parts. The arm lay against the chest, with partial flexion at elbow-, wrist-, and finger-joints. He had no power of movement whatever at the shoulder-, elbow-, and wrist-joints, but he could open and close the fingers slightly. The muscles were flabby and distinctly wasted, and, when tested a day or two later, showed the reactions of degeneration. There was no anæsthesia. No pain or tenderness or other abnormality was detected in the spine. And, in every other respect, at this time the patient appeared to be quite healthy.

About ten days after his admission I first observed a slight want of symmetry in the face. And, a few days later, when this had become more pronounced, I examined him carefully with the following result:—He had slight, but quite unmistakable, paralysis of the left portio dura; he did not close the eye properly, and the angle of the mouth was drawn to the opposite side. The tongue was protruded towards the right, and when at rest in the mouth its right side seemed plumper and occupied a higher level than the other side. The arch of the palate was symmetrical and seemed unaffected, but when phonating the right posterior pillar of the fauces moved inwards to a much less extent than its fellow. There was no affection of the larynx, and I may add that he had no cerebral symptoms, or affection of the eyes or ears. He left on the 9th December, 1889, without any further material change in his symptoms; but he continued to visit me as an out-patient down to the end of January, 1891. During the first month or two little or no change was observed, excepting that the right hand and fingers became congested, swollen and smooth. Subsequently amendment took place, although for a time this was associated with increasing atrophy of muscles, and the development of the characteristic glossy and pointed condition of fingers. Also, but even more slowly, improvement took place in the condition of the face and throat. At the end of February, 1891, I remarked that there was still a trace of weakness in the left portio dura, that the tongue was now protruded straight, that the action of the fauces



was symmetrical, and that excepting for a little wasting of the muscles of the arm generally, imperfect movement of the fingers and almost complete paralysis of the extensors of the thumb, the arm was well. I have no notes of later date, but I saw him occasionally after this, and improvement was still in progress. I must acknowledge that it would probably never have entered into my mind that the symptoms in this case were due to syphilis had it not been for my almost contemporaneous experience of the former case, and for the fact that the lesions here were multiple and successive, and subsided, although slowly, under anti-syphilitic treatment. I think it will be admitted that the earlier symptoms inclusive of the affection of the arm were almost certainly due to meningitis in the neighbourhood of the cervical enlargement of the cord, and that the paralysis of the left portio dura, and the condition of the tongue and throat were dependent on some similar lesion of small extent at the base of the brain.

The case I am now about to quote can hardly be adduced as a proof that syphilitic implication of the brain-substance may under special conditions or at certain stages be undistinguishable from non-specific inflammation of the same part. Moreover, I must admit in regard to it that neither during life nor at the *post-mortem* examination was the question of syphilis seriously entertained. But the case is one of those obscure yet interesting cases which I have pigeon-holed in the hope that something might arise in the course of subsequent experience to throw light upon it. In thinking of it the suspicion of syphilis has been constantly in my mind.

Mary B——, a single woman, aged 32, was admitted under my care on the 27th of February, 1887. She had kept a school, and been in straitened circumstances, and a little before Christmas had begun to feel poorly and suffer from sickness. The sickness had continued, both after and independently of meals, and she had become weak and thin. It was stated that she had lately been indulging in undiluted spirits. She went into St. Thomas's Home, under Dr. Edmunds, on the 8th of February. At that time her chief complaint was of persistent sickness. But, although her intellect appeared to be clear, her manner was strange, and she always repeated questions put to her three or four times before answering. She said that her sight had been failing for the previous two days, and that on the day of admission a loud buzzing

noise in both ears had come on. She had no headache, or paralysis, there was no appearance of thoracic or abdominal disease, and her urine was normal.

The sickness ceased in the course of two or three days, but it was thenceforth very difficult to make her take food. The strangeness of her manner increased, and before long there were marked loss of memory and inability to recognise friends. In addition, her sight became more and more impaired, and on the 18th she could not see persons about her, and asked for a candle in broad daylight. About the same time she complained of unpleasant odours, and on October 24th, it was discovered that she could not smell violets. Her eyes were examined on the 28th. "Right fundus generally hazy, margins of disc ill-defined and streaky, one or two flame-shaped hæmorrhages around it, other hæmorrhages and two or three brightish white dots about the yellow spot, no apparent swelling of disc. Left eye generally in the same condition as the right."

She was transferred to my ward on October 27th. She is a thin woman, making no complaint, lying on her back quietly, or merely picking at the bed-clothes. She answers inconsequently, and often uses a Christian name as of some one of her friends whom she imagines to be present. There is neither anæsthesia nor paralysis, but the limbs are flaccid, and there are no tendon reflexes. She will not open her eyes or mouth, and resists all attempts made to open them by others. No defect of oculo-motor muscles or of pupils. Fundi as before; no definite optic neuritis; and, indeed, as Mr. Nettleship points out, the condition of the fundi is suggestive rather of albuminuric retinitis than of intra-cranial disease. Sordes on lips, tongue coated, pulse 104, small and soft, temperature  $99.8^{\circ}$ , respirations 20, evacuations passed unconsciously. The urine has a sp. gr. of 1024, is free from albumen, but somewhat alkaline, turbid, and presenting crystals of triple phosphate and bacteria.

March 1st. Lies as if asleep, roused with difficulty, breathing stertorous. Eyes widely divergent and slightly turned upwards. Extremities cold, pulse feeble 144, respirations 28, temperature  $98.6^{\circ}$ . In the evening the pulse had risen to 160, and the temperature to  $100.6^{\circ}$ , breath very foul; evacuations passed into the bed. The appearance of the areolæ of the nipples at this time led to a re-examination of the abdomen, when a firm rounded mass



was obscurely felt rising in the mid-line to about 1 inch above the pubes. It was assumed to be an enlarged uterus.

March 2nd, 1.45 A.M. Breathing of the Cheyne-Stokes character, corneal reflexes slight, no twitching or other movement. 10.15 A.M. Quiet, respiration of the same character as before, pulse at times so feeble as not to admit of being counted, but the beats of the heart are 180 in the minute and regular, temperature  $102.4^{\circ}$ , urine alkaline, and containing blood and pus. She died at 9 P.M. For many hours before her death she lay perfectly still, was apparently incapable of being roused, and could not swallow; the divergent squint continued. Her temperature two hours before death was  $104.8^{\circ}$ .

*Autopsy.*—Skull, membranes, venous sinuses, and arteries all healthy. There was no lymph, or tubercle, or undue congestion of surface. In the right ascending parietal convolution just in front of the commencement of the intra-parietal fissure was a round patch of atrophied cortex about as large as a sixpenny piece. There was a slight excess of fluid in the ventricles. The substance of the brain generally was quite healthy. But on careful examination, what was at first supposed to be an infiltrating growth was found to surround the aqueduct of Sylvius, to extend thence upwards so as to involve all but the surface of the tubercula quadrigemina, and forwards so as to include the inner aspect of each optic thalamus as far as the corpora albicantia. In front the affection of the thalami was quite superficial, but on tracing the morbid process backwards these bodies were found more and more encroached on, until at length they were implicated in their whole thickness, and the adjoining parts of the internal capsules were also involved. The soft commissure was included. The lesion was quite symmetrical, and the affected tissues were somewhat swollen, had the same consistency as the healthy brain tissue, presented a pinkish hue, and were studded with punctiform hæmorrhages. Microscopic examination showed the change to be simply inflammatory. The bladder was dilated, hypertrophied and injected; the pelves of the kidneys were also congested and contained a small quantity of pus-like fluid. The kidney structure was healthy. The uterus contained a three-months fœtus, and presented a few small fibromata. All other organs were healthy.

My reasons for suspecting syphilis in this case were the facts of the patient's immorality and the discovery of an old area of



degeneration in the brain, and the difficulty of accounting otherwise than by syphilis for the curious lesion which caused her fatal illness.

Having now brought before you all the cases of fatal typical or suspected syphilitic disease of cerebral arteries, of intra-cranial gummata, and of syphilitic inflammation of the membranes or substance of the brain, which I had selected for the purpose, it may be convenient to consider, however briefly, the subject of the differential diagnosis of these several lesions. Judging from the analogies afforded by embolic obstruction of cerebral arteries, it might not unreasonably be supposed that obstruction of vessels due to syphilis would be indicated by paralytic symptoms of sudden onset; while, on the other hand, we know that the growth of tumours is, as a rule, revealed by the gradual development of headache, giddiness, and sickness, the specific signs usually being evolved at some later period; and hence it might seem not unlikely that we have a ready means of differential diagnosis. But on the other hand, so far as I know without exception, in all the cases of syphilitic obstruction of arteries that have come under my immediate observation, the onset of symptoms has been gradual, there has been, before the occurrence of paralysis, a prodromal period of headache and sickness, or other morbid signs, due doubtless to the inflammatory changes going on in the blood vessels themselves or in their immediate neighbourhood; and further, in no inconsiderable number of cases a brain tumour goes on growing without causing any appreciable symptoms, until suddenly the patient has an epileptic fit or some other attack which forms the clinical starting point of his malady. Again, it must not be forgotten that gummata, pachymeningitis, and disease of arteries are often of simultaneous development. Nevertheless, it may be admitted that if, in the course of vague cerebral symptoms a syphilitic patient suddenly has some kind of seizure and becomes hemiplegic, the probability is that he has obstruction of vessels with the softening or hæmorrhage, which such obstruction is liable to induce; and, on the other hand, it may equally be admitted that if such a patient suffers from severe protracted cerebral symptoms, such as headache, giddiness, vomiting, optic neuritis, mental perversion or failure, and occasional or periodical epileptic attacks, the probability is that he is the subject of gummata or syphilitic pachymeningitis—a diagnosis the prob-

ability of which would be increased, if at the same time there was evidence of syphilitic disease of the periosteum of the skull or of the ears. Again, gummata and pachymeningitis are specially apt to affect the parts at the base of the brain, and to implicate nerves; and hence the association of even vague cerebral symptoms with progressive and disorderly implication of the cerebral nerves points to one or other or both of these affections; and in fact there is no doubt that we are most likely to meet with hemiplegia in connection with obstruction of vessels, and implication of nerves in connection with gummatous tumour or infiltration. It must not be forgotten, however, that syphilitic obstruction of vessels is not limited to the middle cerebrals as embolic obstruction for the most part is, but that any of the arteries may suffer, and that as a consequence we may get softening of the pons or crura cerebri, and together with more or less hemiplegia implication of many nerves, a condition closely resembling the consequences of basal gummata. On the whole I should be disposed to say that, having regard to the mode of invasion, to the character and frequency of epileptiform attacks, to the fact of the sudden or gradual development of hemiplegia, to the grouping and evolution of paralyses of cerebral nerves, we may in many cases form a reasonably accurate differential diagnosis, but that in the majority of cases no such accuracy is attainable, and, I may add, is unimportant.

I propose now to bring before you, as clinical studies, two or three cases presenting points of special interest, but in which the diagnosis has not been put to the test of *post-mortem* examination.

The first is a case which I had the privilege of seeing on several occasions with Dr. C. D. F. Phillips. The following is in effect the note which I made on the occasion of my first visit, on the 1st November, 1892. "The patient is an unmarried man, aged 42, who had primary syphilis about twenty years ago, followed in due course by sore throat, cutaneous rash, and loss of hair, but he made an apparently good recovery, and remained free from specific symptoms for many years. Three or four years ago he had a rash which he thought was syphilitic, and about the same time he seems to have had a mild attack of influenza. He dates his present illness from the beginning of the year. He then had what he regarded as a recurrence of influenza, which was followed by



occasional attacks of giddiness, and by some defect of sight. The latter was attributed by an oculist, whom he consulted, to astigmatism, but he himself noticed that he did not see, or failed to observe, things to his right side, and that thus he was apt to overlook friends and acquaintances passing him in the street. These symptoms have continued, but during the last few months others have accrued, and he has been getting seriously ill. He has lost flesh and strength, and become low-spirited. He suffered for some time from sickness in the morning, and occasionally at other times of the day. But he has been better in this respect of late. A month or two ago he became deaf in the left ear; this improved in a short time, and then the right ear became similarly affected. Later the left ear became again the worse of the two. But he never had earache or otorrhœa. For some few weeks he has complained of a sense of coldness and numbness throughout the whole of the right half of the body, of pins and needles in the right hand, of a feeling of insecurity in walking and a tendency to fall over or stagger to the right, and of clumsiness in the use of the right hand. His speech seems to have been getting thick for the last few weeks, and he states that he has occasionally seen double. Has never had fits. He is thin, weak, pale, and looks ill. He is much depressed and nervous about himself, but quite sensible. He complains still of the coldness and numbness of the right side, of the tingling in the hand, and of some difficulty in the free use of the arm and leg; and when trying to walk I observe a little tendency to stagger and to fall over to the right side, but he can feel (apparently perfectly) everywhere, and there is no obvious impairment of strength in either of the affected limbs; moreover, he can stand steadily with eyes shut, and the reflexes are all normal. There is general but incomplete paralysis of the left side of the face; he cannot close the eye or wrinkle the forehead, and his mouth is drawn to the right. There is no affection of the tongue or palate. He talks thickly, slowly, and with some hesitation, and I think there is a little tremor of the lips; phonation is perfect. The left pupil is smaller than the right, but both pupils act to light and accommodation; he states, however, that of late he has lost the power of reading print at a medium distance from his eyes, and of readily accommodating for objects varying in position. He cannot follow with his eyes; he cannot move either of them to the left beyond the mesial line, and can move them



only half way towards the right canthi, and this only with much effort and with the production of nystagmus. He can move the eyes up and down readily, but nystagmus attends the upward movement. No ptosis. On rough examination there seems to be no contraction of the fields of vision. The fundi are normal. He denies seeing double now, but the eyes do not converge on the object at which he is presumed to be looking. He is deaf with both ears, especially with the left, with which he cannot hear a watch in contact with the pinna. He can, however, hear a loud-ticking clock with this ear, if it be applied to the ear itself or to the head. He has loud noises in both ears, and complains of a sense of tightness in the head, but not of actual pain. There is no evidence of disease in either the thoracic or the abdominal viscera."

I saw him again on the 15th, when, in his general condition and in one or two minor points, there seemed a little improvement. But he still complained of coldness of the right half of the body, of pins and needles in the right hand, and clumsiness in using it, and of tendency to fall over towards the right. The paralysis of the left portio dura was complete, and the condition of the eyes and ears remained unaltered. His speech, however, was more indistinct, and he said that the left half of the tongue and lips felt cold.

I saw him for the last time on the 26th. He was then not nearly so well as he had been. He had become very weak and intensely depressed, and had kept his bed for several days. His right arm and leg were distinctly weaker than their fellows, and the grasp of the right hand was markedly feeble. The paralysis of the left portio dura continued. His speech had become almost unintelligible, and he could only utter two or three words on a breath, but phonation was unimpaired. He had had some difficulty in masticating and swallowing, and had been compelled to have his food pounded, in which state he could take it without much difficulty; but drinking made him choke. He could not open his mouth wide, or put out his tongue well. The latter did not, however, present any traces of unilateral paralysis, nor did the soft palate. On asking him to close his jaws firmly scarcely any contraction could be felt either in the temporal muscles or in the masseters; and it appeared that for some days supporting his chin with his hand had been of assistance to him both in masticating and in speaking. The pupils were small, equal, and acted to light.

The eyes moved freely upwards and downwards, but there was now no lateral movement whatever. The deafness continued. The thoracic muscles and the diaphragm acted, but I thought they were enfeebled. The more noteworthy changes for the worse that had taken place were the development of right-sided palsy, the weakening of the muscles supplied by the motor-branches of the 5th pair, the increase of the paresis in the lips, tongue, and muscles of deglutition producing symptoms like those of glosso-labio-laryngeal palsy, and the supervention of shortness of breath, and absolute loss of power in all the muscles causing lateral movements of the eyes.

He continued to grow worse, and in the early part of December, while in the act of drinking, died suddenly apparently from laryngeal spasm.

There is very little reason to doubt that the above was a case of cerebral syphilis. But what was the exact nature of the lesion present and where was it? I will endeavour to answer this question by narrating another case, which, by-the-bye, may furnish an argument against my syphilitic hypothesis. The case is one of which I published the full details a year or two ago in 'Brain.' A man, aged 49, came under my care on March 12th, 1890. He had been a heavy drinker, but denied having had syphilis. His illness began seven weeks previously with a noise in his ears as of bellows blowing, and numbness of the left thigh and coldness of the left shoulder. Since then the noise has become constant, mainly in the left ear; he has suffered from headache referred to the back of the eyes; he has complained of giddiness and inability to walk straight; he has occasionally seen double; he has lost the power of biting on the right side, and his articulation has become impaired; paralytic weakness of the left side has come on; and he has been troubled with cough.

On admission he complained of the above symptoms. The right masseter and temporal muscles were completely inactive so that he could not masticate or bite with firmness on that side. There was very marked impairment of sensation over the upper half of the right side of the face, and over the whole of the left half of the face and head and neck. He could move his head freely, but there was conjugate deviation of the eyes to the left. The pupils, of which the left was the larger, acted to light and accommodation; the fundi were normal, and, excepting that he had double vision for all distant objects, his sight seemed good. Hearing was im-



paired, especially in the left ear. The sense of smell was normal; but sensation was much blunted, not only in the lips but also throughout the whole of the interior of the mouth and in the tongue, and he distinguished flavours with difficulty. There was no paralysis of the tongue, and the soft palate was symmetrical, but it was motionless during articulation and displayed no reflex movements. His speech was indistinct, but his voice had tone. The left arm and leg were markedly enfeebled, and there was almost complete anæsthesia throughout the left half of the body to the middle line. The reflexes on both sides were normal. He remained in the hospital until his death which took place on the 4th July. During this period various additional symptoms and complications arose to which I need not call attention. But some directly due to the progress of the cerebral disease and bearing on its diagnosis call for consideration. Thus he had at times difficulty in swallowing and was apt to choke; paralysis of the right portio dura became developed; paralysis of the right arm and leg came on, but was never so complete as that of the left side, and was apparently unattended with anæsthesia; and further about the middle of April it was observed for the first time that the conjugate deviation of the eyes had disappeared and that he looked straight forward. Careful examination showed that he could not move either eye to the right or left, but that he could move them upwards and downwards with perfect ease. There was never any optic neuritis. He died at last from gradually increasing weakness, with accumulation of mucus in the bronchial tubes due to intercurrent bronchitis.

It is obvious that, while there was a good deal of difference in details, there was a striking resemblance in many important particulars between these two cases. In both there was hemiplegia beginning with a sense of numbness and coldness; in both there was paralysis of the 7th nerve of the opposite side; in both there was implication of the 5th pair; in both there was deafness, attended with loud noises in the ears; in both there was difficulty of speech, mastication, and deglutition; in both there was a peculiar and striking form of ophthalmoplegia externa; and in both there was absence of fits and of optic neuritis. In the latter case, I assumed during life that the patient was suffering either from softening of the pons connected with diseased and obstructed vessels, or from a tumour in the same situation. The question of



syphilis was in my mind, but I did not venture upon any decided opinion on the subject. The *post-mortem* revealed in the substance of the pons a cheesy tumour, about as large as a good-sized chestnut, surrounded on every side by a thin layer of nervous substance, and embedded in the surface of one of the lobes of the cerebellum another mass as large as a hazel nut, which presented a dense, fibrous capsule about half a line thick, and of which the interior consisted of cheesy matter, becoming dry, cretaceous, and friable. There was no further disease of the brain, and the vessels were healthy. In the apex of the left lung were some indurated patches and scars, obviously due to old tubercular disease. The masses in the brain were tubercular. An interesting point in the case is that the tubercular tumour in the cerebellum must have been of old date, like the scars in the lungs, and that there was no history of symptoms due to its presence. With the incidents of this latter case before us, I think it may be safely assumed that the intra-cranial disease in Dr. Phillips's case involved mainly, if not exclusively, the pons Varolii, and that it was either a large gumma, or a patch of softening connected with obstructed arteries.

Another case which was of exceeding interest to me came under my care on the 17th January, 1883. It was published by me in a paper entitled "Cases of Recovery from Symptoms pointing to the Presence of Progressive Organic Cerebral Disease," which appeared in 'Brain,' for April, 1885. Its chief interest lies in the fact of practically complete recovery from ingravescient symptoms of a very grave and threatening character.

The patient was a single woman, a nurse, aged 39. She had had good health, and there was no history or evidence of syphilis. She had been attending on a private patient, when suddenly, a month ago, she was attacked with giddiness, nausea, and double vision. These symptoms continued, and three weeks later she found on getting out of bed that she could not stand. On the 12th January she first complained of headache in the frontal and occipital regions; and on the 15th she vomited, and noticed numbness and weakness in the right half of the upper lip.

She is healthy-looking, but complains of headache, nausea, giddiness, with consequent inability to stand, and difficulty in using the right half of the upper lip. The headache is severe, and mostly frontal; but there is an area of tenderness to percussion at the back of the left parietal bone. The forehead also is somewhat

tender. The nausea, which is distressing, comes on mostly when she sits up or stands. She cannot walk or even stand without assistance, staggers like a drunken person, and has a tendency (she says) to fall to the right side. There are no ataxic movements. There is slight, but obvious paralysis of the right facial nerve; the right eyelids close imperfectly; the right upper lip clearly acts feebly; and the right side of the face is evidently smoother than the other; but the right angle of the mouth moves freely when she laughs. The tongue is protruded slightly to the left. There is no obvious difference in the appearance or action of the two sides of the soft palate, but the uvula is concave, and its apex points towards the right. The right external rectus is completely, the left internal rectus partly, paralysed; and there is well-marked horizontal nystagmus when she looks strongly to the left. Pupils normal; no optic neuritis. There is no further evidence of disease. Repeated examination during the next few days confirmed the accuracy of the account above given; but Mr. Nettleship found out additionally that the right half of the field of vision for both eyes was so largely contracted as almost to constitute hemianopsia. On the 23rd it was noticed that there was weakness of the left external rectus, and this became more pronounced during the next four days. On the 27th she first experienced a feeling of numbness and coldness at the bottom of both feet, which in the course of a few days amounted to a sense of pins and needles. On February 12th she complained that her legs felt stiff, and that they jumped at times. She could move her legs pretty freely, but they tended to become stiff, especially at the ankle and knee joints; there was marked exaggeration of the tendon reflexes, and on the left side ankle clonus. The phenomena continued for a time, the left leg being worse than the other. Ankle clonus was obtained somewhat later on the right side. On February 3rd, it was noticed that the facial paralysis had increased, and also that she had frequent twitchings of the left angle of the mouth; and on the 20th (these symptoms continuing) it was noticed that she had occasional twitchings of the left eyelid, and slight tremors in the left hand. She had been complaining for some days that her eyesight had been getting indistinct; and an examination made on the 1st March showed that, while the ocular paralysis and field of vision remained as before, her discs were somewhat pale, and she was colour-blind.



She could not distinguish greens or reds, and confounded them with brown and grey. Bright-yellow she called white, and bright-blue and lilac both dark-blue. On March 30th, after suffering for a few days from much more intense headache than usual, she for the first time complained of a feeling of pins and needles in the right hand and arm, and of pain in the right shoulder. The arm also became weak, and in the course of a week or two slightly flexed at the several joints, the fingers especially suffering, and the pain extended from the shoulder downwards. The paralysis was never complete, but attained its maximum towards the end of April. During the greater part of the time terminating with the last date the patient had been getting slowly but steadily worse; the pain in the head (which had varied in position, but become more and more localised as time went on about the back of the left parietal bone) had been constant, but liable to severe exacerbations; the feeling of nausea on the slightest movement had continued; and from time to time (as has been shown) additional paralytic phenomena had arisen. Nevertheless, during April, some favourable indications were noticed. On the 11th she saw single for the first time, and on the 15th it was noticed that the movements of the eyes were perfect, and that she was recovering her colour vision. Early in May she began to improve decidedly. She still suffered from headache, giddiness, and nausea, but the attacks were less frequent. About the 10th she began to sit up, and a week or two later to walk with assistance. I need only add, that when she left the hospital, on June 4th, there was scarcely a trace of headache or nausea; the facial nerve was not obviously paralysed; her visual functions were perfect; the right hand and arm had almost completely regained power; there had been no startings in the legs for weeks, and she could walk well. She went for a month to a convalescent home, and on her return appeared to be, and expressed herself as feeling, quite well. I saw her some months afterwards, and she continued well.

What the nature of the case was remains unsettled. The question as to whether it was a functional or hysterical disorder was fully considered and rejected. But nevertheless that rejected view may have been the true one. On the other hand, the curiously ingravescent progress of the disease, involving hemiplegic symptoms, paraplegic symptoms, paralysis of various nerves at the base of the brain, and visual troubles, undoubtedly suggested some pro-



gressive lesion. Moreover, she recovered under the persistent use of iodide of potassium and mercury.

Syphilitic affections of the spinal cord or its membranes are by no means uncommon; but I have to confess that although I have naturally seen many cases of what I have regarded as syphilitic paraplegia and some which have proved fatal, I have never had the privilege of following one of these latter to the *post-mortem* room, so that consequently my personal experience of the morbid anatomy of such cases is very deficient. There is abundant proof, however, that within the spinal canal as within the skull, syphilitic arteritis, gummata, and syphilitic pachymeningitis are all liable to occur. I propose to quote briefly three cases each of which presents some point or points of special interest.

The first is that of a girl, aged 21, who came under my care on the 7th June, 1881. There was no history of chancre, but in the preceding August she first noticed a scaly eruption which increased and became general during the next few months; and at Christmas she became a patient of Mr. Nettleship's for an attack of syphilitic iritis. At this time the rash was still out. She was treated specifically with benefit; but in May some large hard glands appeared in the neck and at the end of the month she began to complain of pain round the waist and down the legs, worse at night-time, of difficulty in walking, and of incontinence of urine. On admission a week later, she was healthy-looking, but still presented traces of her rash. The iritis had subsided. She had no voluntary power whatever over the right lower extremity, and could move the left only very slightly. There was no anæsthesia, but she had pain in her legs. The superficial and tendon reflexes were all very brisk, and cloni were readily obtainable. She had incontinence of urine. There was no pain or tenderness in the course of the spine and no sign of disease in any other organ. She remained in the hospital six months, during which time (after a period of hesitation) her paralytic symptoms slowly improved, and at the time of her discharge, although the legs were still somewhat weak and dragged, she could walk without assistance, and she had fairly well recovered power over the bladder. Her progress was retarded by the occurrence of cystitis and the formation of bed-sores. Her treatment, both by Mr. Nettleship and while under my care was strictly anti-syphilitic. The points of chief interest are that when the paraplegia appeared the patient was

still suffering from secondary symptoms, and that she had been under the influence of specific treatment for about five months.

My second case is that of a young man, aged 31, who was admitted under my care on the 19th June, 1891. He had had a chancre in 1881, followed by an eruption. His present illness began in January with pain extending down the right arm. After this had continued for three weeks he observed that the limb was becoming weak and thin. These symptoms continued; and three weeks ago he was attacked with pain in the right leg, which was followed in the course of two or three days by pain in the left leg, and loss of feeling in both legs. On June 3rd his water began to run away from him, and three days before admission this condition was replaced by retention. His bowels had been constipated.

He was healthy-looking. He had no cerebral symptoms, and no tenderness in the course of the spine. The right trapezius, supra-spinatus, infra-spinatus, deltoid, triceps, biceps, and supinator longus were all much wasted, and the arm and forearm were from  $1\frac{1}{4}$  to 2 inches less in girth than the left. The movements were all feeble. There was absolute loss of voluntary power in both legs, with exaggerated tendon and superficial reflexes; no tactile anæsthesia, but entire loss of sense of pain. There were pigmented scars on the legs, and extensive scarring on both shoulders behind. No further evidence of disease.

On the 23rd it was noted, that during the previous night "he had become rigid, and his speech indistinct; that in the morning this indistinctness of utterance was still present; that his left arm and the lower part of the left side of the face were paralysed; that he had some difficulty in swallowing solids, and that he passed his motions involuntarily."

During the next few days the loss of power in the left arm increased, his legs became quite anæsthetic in their whole length: he had constant jerkings in them, and he suffered from pain referred to both temporo-maxillary articulations, to the muscles of the left arm and to the sternum. Subsequently there was some improvement as regards the movements of the left arm and the sensibility of the legs; but bed-sores formed, his urine became alkaline and offensive, and finally hypostatic congestion of the lungs supervened, and the case ended fatally. No *post-mortem* was allowed. It may be added that Mr. Lawford, who examined the eyes for me, reported the discs pale, filled in, with diminished



arteries; a little disturbance of choroid at outer side of right—probably post-syphilitic atrophy.

The interesting features in this case are, that the disease obviously began with gummata or inflammation of the membranes of the cord about the cervical enlargement, the cord itself becoming involved later, and that syphilitic disease of the brain supervened. The case, indeed, reminds me of the second case which I placed before you this evening, in which (although there was neither hemiplegia nor paraplegia) there was evidence of pachymeningitis about the cervical enlargement, and of the super-vention later of similar disease at the base of the brain.

The third case is one that has recently been under the care of my colleague, Dr. Payne. The patient was a painter's labourer, aged 24. When 16, he was crushed between a railway carriage and the platform, as a result of which, it is said, he had complete paralysis of both legs and weakness of both arms. He had recovered at the end of eleven weeks, and was able to resume work; and he continued well until eighteen months ago. About that time he had enlargement of the left testicle, which became as "big as his fist." An abscess also formed between the testicles, which was ruptured by accident and soon healed. He was under treatment for six or seven weeks, and took medicine which made his teeth sore. About four months afterwards he began to complain of pain on the left side of the head from the occiput to the eye, and of giddiness and nausea; and he became deaf in his left ear, and his left upper eye-lid drooped; but his sight was unaffected, and he did not see double. For six weeks he was under medical treatment for these symptoms, and it was while he was under treatment that his legs became weak. The headache, giddiness, and drooping of the lid gradually subsided, but the paralytic symptoms slowly increased upon him, until, four months ago, he had to give up work. At this time he was markedly unsteady in his gait, his left hand was clumsy, he complained that his left hand and leg felt numb and cold, and he had recurrence of cephalalgia, severe shooting pains in the thighs and legs, and incontinence of urine. Eleven days before admission he complained of a mist before his right eye, which gradually increased and became attended with pain. He has never had headache; nor does he allow that he has ever had syphilis.

He is a robust-looking man. There is marked incoordination



of movement in both lower extremities. He stumbles as he walks, especially when turning round, and he cannot stand with eyes closed. He has impairment of sensation in the left leg from the knee downwards and in the sole of the right foot, and when walking feels as if he were treading on something soft. Knee jerks and plantar reflexes absent. The muscles are not wasted and have considerable power. He has shooting pains in both legs, but mainly in the left. He has numbness in the tips of the fingers of the left hand, and the movements of the part are incoordinate and clumsy. The conjunctiva of the right eye is deeply injected and discharges muco-pus. The cornea is ground-glass-like, especially in its upper and inner part. There is no sign of ptosis, and the movements of the eyes are perfect. The pupils are equal, and act to light and accommodation. The left ear is somewhat deaf. The teeth are normal. He can micturate voluntarily; but when the bladder contains much water, this tends to trickle away. He improved under the use of iodide of potassium and mercury, and when I saw him seven weeks after admission his walking power had much improved, but he was still ataxic and unsteady and incapable of walking without assistance; he had quite recovered the use of his left arm; the numbness had disappeared from the left hand and right foot, and remained only in the course of the left shin; he had lost his headache and neuralgia; his eye had greatly improved; he was still deaf, and his knee jerks were absent. His left testicle was larger than the right, and its epididymis was large, nodulated, and hard.

This case was interesting partly because the question arose as to whether the patient was suffering from acquired or congenital syphilis; partly because, as in the last case, paraplegic symptoms were associated with symptoms referable to intra-cranial mischief; but chiefly because the symptoms, so far at any rate as the lower extremities were concerned, presented the characteristic features of locomotor ataxy. I feel no doubt myself that the syphilis was acquired; and that the ataxic symptoms were due to specific syphilitic disease involving mainly the posterior columns of the cord. I do not think that the railway accident or lead-poisoning had anything to do with his condition.

Of syphilitic affection of the nerves, apart from those at the base of the brain and in the spinal canal, I have no knowledge or experience. But it seems not unreasonable to assume that nerves

in any part of their peripheral course may not only become involved in syphilitic lesions originating in their neighbourhood, but also may be the seat of independent gummatous infiltration of limited extent. Such cases have been described; and are probably not altogether uncommon. And at any rate localised anæsthesia, neuralgia, or paralysis occurring in syphilitic persons, and not due to cerebral disease might reasonably be suspected of such an origin and be treated accordingly. Symmetrical peripheal neuritis, however, occurring in a syphilitic patient I should certainly be indisposed to regard as syphilitic excepting in that vague sense in which many constitutional symptoms following on syphilis are regarded as parts of that disorder. I do not purpose to discuss the symptoms due to implication of each of the intra-cranial nerves. But I shall content myself with making a few remarks on affections of the nerves supplying the muscles of the eyeballs, and I shall do this because some of my cases are interesting in this relation. Total and complete ophthalmoplegia externa I have never met with excepting in cases of functional nervous disorder. In one such case it was associated with many other paralytic symptoms, continued for two years, and when at the end of that time the patient died from an accidental intercurrent disorder, no trace whatever of disease was detected in any part of the nervous system. In another such case in which I watched the gradual evolution of the ophthalmoplegia, and in which also there were many other grave symptoms present, the ophthalmoplegia lasted for nearly the same length of time as in the former case, and then the patient recovered completely. Conjugate deviation of the eyes, presumably due to some lesion involving the nucleus of origin of one of the 6th nerves or the strand of fibres passing thence upwards to the opposite hemisphere of the brain, occurred in several of the cases which I have adduced as actual or suspected cases of cerebral syphilis. The occurrence of this form of ophthalmoplegia, like that of the total ophthalmoplegia, could scarcely be produced by any lesion excepting one situated within the pons or some other part of the brain. It is curious that in two of my cases there was what I ventured on one occasion to call, or rather miscall, double conjugate deviation. It will be recollected that in the case of tubercle of the pons, which I quoted earlier in this lecture, one of the early symptoms was conjugate deviation of the eyes to one side, and that after a time the conjugate deviation suddenly ceased, and that thenceforth the patient had no



power of moving the eyes to the right or left, though retaining the power of moving them upwards and downwards freely. It seemed as though the condition originally causing deviation in the one direction had become complicated by a condition tending to cause deviation in the opposite direction, and that these, so to speak, balanced one another. It is probable that first one-sixth nucleus became affected, and then the other. The same phenomenon was present in Dr. Phillips's case, and helped to make me believe that the patient's lesion was in the pons and not meningeal or simply nervous. On the last occasion on which I saw this patient he was looking straight in front of him, and while he could not move either eye in the slightest degree to the right or left, his vertical movements were perfectly free. I may recall the patient's statement that in the earlier part of his illness he did not notice persons or things to his right side, and yet he had not when I saw him, and seems never to have had, hemianopsia. It has seemed to me probable that at that time he had a tendency to conjugate deviation of the eyes to the left, and that this was the cause of his visual default. There is no doubt that in syphilis the motor nerves of the eyes are peculiarly prone to suffer, and that not unfrequently all of them become involved in a greater or less degree. But in my experience the paralysis always remains incomplete or unsymmetrical.

I had intended to say much more than I have done concerning syphilitic affections of the nerves, and to add a few remarks in respect of the treatment of syphilis. But, gentlemen, I have already occupied the whole of the time you have placed at my disposal; and I do not feel justified in further encroaching on your kindness, the more especially as there is nothing in what I had intended to say on either of these topics that is particularly novel or particularly interesting.

Here, then, I conclude my Lettsomian Lectures, and thank you, Mr. President and gentlemen, for the great kindness and patience with which from first to last you have listened to them.

---



*February 13th, 1893.*

# ON THE PREVENTION OF SHORTENING AND OTHER FORMS OF MAL-UNION AFTER FRACTURE, BY THE USE OF METAL PINS PASSED INTO THE FRAGMENTS SUBCUTANEOUSLY.

By C. B. KEETLEY, F.R.C.S. Eng.

THE object of this paper is to demonstrate a method of preventing or of removing shortening after fracture of the long bones by the use, combined with simple external appliances, of rigid metallic pins, inserted subcutaneously, or, at least, without a cutting operation. Oblique fractures of the femur are those which most commonly unite with shortening. I have had no opportunity of applying to any other bone the method about to be described, and the femur is an excellent type of all long bones; it will be convenient, therefore, until the final paragraphs of this paper to consider the femur only.

It is well known that surgeons of great eminence and high character have claimed uniform success in treating fractures of the femur without resultant shortening; but I think the trenchant criticism of these claims given in "Hamilton's Treatise on Fractures," proves conclusively that they have not been made out, and are not to be trusted. Hamilton devotes ten pages to this criticism, which shows the great importance he attaches to the question. The latest important article on fractures—namely, that in the second volume of Duplay and Reclus' great 'Traité de Chirurgie'—quotes Hamilton with approval on this point, and adds nothing of importance. I will venture to take it for granted that serious shortening occasionally follows fracture of the femur treated, however carefully, by all methods hitherto described. On the question of amount of shortening, Hamilton sums up as follows:—"When, in consequence of displacement, an overlapping occurs, the average shortening of simple fractures in adults, where the best appliances and the utmost skill have been employed, is from one-half to three-quarters of an inch"; but he does not deny that measurement of museum specimens gave a less favourable

result. Of nineteen specimens in Mütter's cabinet, not one seemed to him to be shortened less than an inch; indeed, one showed a shortening of  $2\frac{1}{4}$  inches and one of no less than  $3\frac{1}{2}$  or 4 inches. Both these were fractures of the middle third. Surgeons who have gone on obtaining nothing but reasonably satisfactory results for years should give some of the credit to fortune as well as to their own skill. Even their less successful or more severely self-criticising brethren do not meet with extreme cases every day. Next, with regard to the effect of shortening. Though it be true that patients with an inch of shortening may get on very well, and with little or no limp, let anyone disposed to make light of such a misfortune spend some morning walking about with one boot on and the other off, and then say how he would like to be condemned to do so for life. As a matter of fact, all except careless or vain people, with even no more than  $\frac{3}{4}$ -inch shortening, wear a surgical boot raised by cork, and this means a serious and ceaseless expense to persons of small incomes. Moreover, no raised boot really corrects the deformity; it compensates for a short thigh by making a long leg and raises the knee-joint and ankle-joint to an abnormal situation.

The following conclusions are to be deduced. A not inconsiderable proportion of cases of one of the commonest varieties of fracture known have united with serious shortening in spite of the careful use of the various appliances hitherto described. In 1893 the case stands very much as it did in 1801, when Benjamin Bell wrote:—"An effectual method of securing oblique fractures in the bones of the extremities, and especially of the thigh bone, is, perhaps, one of the greatest desiderata of modern surgery. In all ages the difficulty of this has been confessedly great; and frequent lameness produced by shortened limbs arising from this cause evidently shows that we are still deficient in this branch of practice."\* But the general position in which surgeons stand now is one of great advantage compared with that of the time of Benjamin Bell, for there are now anæsthesia, antisepticism, and subcutaneous surgery. Benjamin Bell, keen-sighted as he was, had to survey his field of action from a molehill; the present-day surgeon looks at his from a mountain top, from a height which, I think, brings into view a path surmounting the difficulties of

\* B. Bell's 'System of Surgery,' vol. vii, p. 21, quoted by Hamilton.

treating oblique fractures of the femur. I am not going to advocate open incision, with pinning, pegging, or wiring the fragments together. Experience and the records of such treatment when applied to ununited fractures make it probable that such a plan, if at all generally adopted, would lead too often to failure of anti-septic precautions and consequent calamities. On the other hand, there are strong reasons for thinking that a carefully purified pin of thickly plated steel, if made to enter through a puncture in the skin, cleansed with equal care, can be passed through muscle and bone and left there for a considerable time with impunity. Assuming two such pins to be used, one perforating the upper, and the other the lower, fragment of a broken bone, there are also powerful *a priori* reasons for thinking that these two pins could be held at a right and reasonable distance apart also with safety. Moreover, the use of these pins would not prevent the simultaneous employment of splints, cradles, extensions, or other external appliances used in the ordinary manner. It is easy to see that these principles could be applied in a great variety of ways. For example (1) the pins could be made to perforate the limb completely, or to stop short on one side of the bone; (2) they could be sharp, or screw-pointed, or gimlet-pointed, and could either be made to bore their own way, or a passage could be prepared for them by an independent instrument; (3) when *in situ* they could be held apart in different ways, too many even for enumeration; (4) only one pin might be used, the fragment under most control (usually the distal one) being held in place and at proper distance by a wholly external appliance reacting against the solitary pin passed through the other fragment; or the pin might be passed through both fragments like a rivet, as was recommended by Gaillard; (5) the separation force could be rigid or elastic, or a weight extension; and (6) as any method of using pins could be combined with almost any kind of external appliance in use, the possible combinations to choose from are enough for nearly every surgeon to have a method of his own. The details of the method I have myself used and now recommend, are as follows. The instruments required are: (1) two L-shaped pins of hardened steel thickly plated with silver; (2) the patella instruments described by me in the 'Lancet' for December 10th, 1892, or, at least, the long-bladed bradawl; (3) a pointed tenotomy knife; and (4) a flat sand pillow. Each L-shaped pin consists, of course, of two arms, one cylindrical (AA'), the other (BB')



flattened in a plane at right angles to the rounded arm. The cylindrical arm is of the diameter of a No. 7 (French scale) catheter—*i.e.*, it is 7 millimetres in circumference—and has a

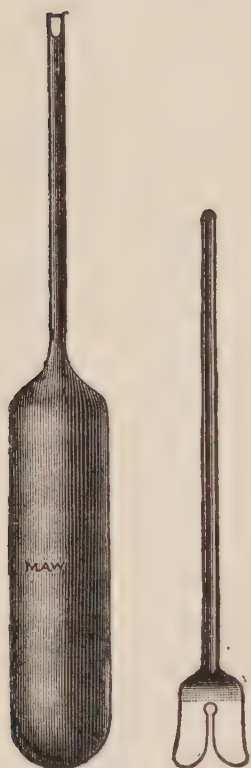


FIG. 1.

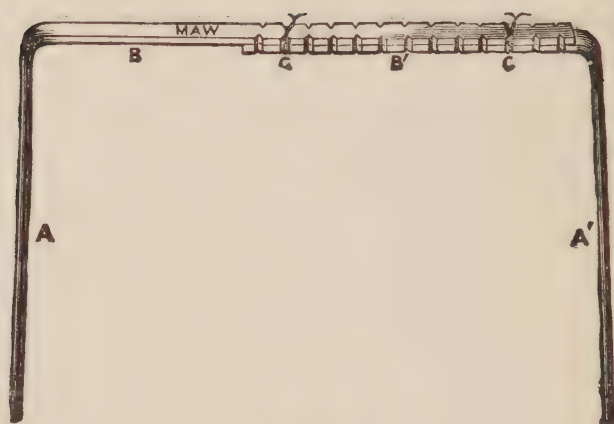


FIG. 2.

rounded blunt point. The length is  $3\frac{1}{2}$  inches, *i.e.*, nearly 9 centimetres. The flattened arm of one pin is 3 inches long, that of the other being 5 inches. Both are  $\frac{1}{4}$ -inch wide, about  $\frac{1}{10}$ -inch thick, and there are notches at the edges at intervals of  $\frac{1}{4}$  inch. The patella bradawl has, it may be mentioned, a blade  $3\frac{1}{2}$  inches long, and both its blade and its handle are of one smooth piece of nickelled steel, the handle being made hollow for lightness.

*Operation.*—Stringent antiseptic precautions must be used, and they are particularly easy in an operation like this, where the open wound is a puncture, and nothing enters it except two or three straight and smooth pieces of plated steel. This easiness is no excuse for carelessness—it only makes neglect inexcusable. The following are the details of the operation:—1. Reduce the fracture under ether, breaking it down again if union has taken place recently in the bad position. 2. Whilst two assistants make extension and counter-extension, adjust the fractured ends and try

to ascertain the precise seat and place of fracture. Select a point in each fragment likely to be clear of the fracture, and in such a position that a straight line from it transversely perforating the bone would keep well clear of important vessels and nerves, synovial membranes, &c. In both my two cases I have been surprised to find how far it was necessary to go from the fracture in order to find a firm place in the lower fragment. The two points selected should lie as nearly as possible above one another, and not in a spiral line circling the limb. 3. Puncture the skin, push the bradawl through the puncture down to the bone, use it very gently as a probe to define the position of the bone, and then bore steadily through. Take care not to break the long blade and not to perforate obliquely. 4. Slip the director along the awl. If the handle of the latter is in the way bend the former a little and have the skin pressed down on the bone. 5. Withdraw the awl, twisting and not jerking it out; keep the director carefully in position. 6. Pass in one of the pins through the bone and withdraw the director. Repeat 3, 4, 5, and 6 in the case of the upper fragment. Take care to bore both fragments in the same longitudinal plane. 7. The two pins being now in the bone, turn them into such a position that the flat arm of one lies upon and touches the flat

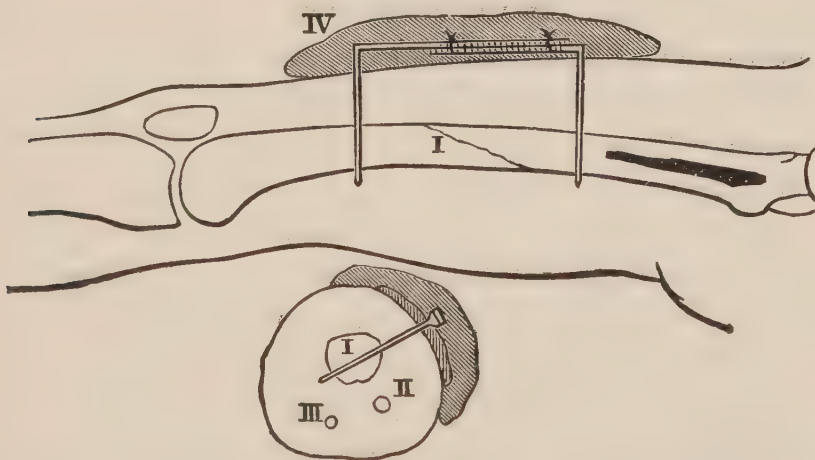


FIG. 3.—I. Femur (lesser fragment). II. Artery. III. External popliteal nerve. IV. Dressings.

arm of the other. If the pins are to remain less than 5 inches apart, the long-armed pin must lie over the other. The advantage of making the flat arms of different lengths will now be seen. The pins can be thereby fixed at any distance from 2 to 6 inches

apart. 8. Full extension being made, and the limb demonstrated by measurement to be of proper length, lash the flat arms of the pins together with silver wire pulled taut and twisted home at the ends. See that there is full tension on the pins before fixing them, for they have a slight elasticity. If this point is not attended to  $\frac{1}{4}$  inch or more may be lost.

*Dressings.*—Iodoform gauze wrung out in sublimate solution (1 in 2,000) should be placed beneath and above the external arms of the pins; the gauze should be flat and not lumpy. Over the gauze a well-rolled wood-wool pad should be placed, and all round the limb at and near the fracture there should be a sheet of absorbent wool. The whole should be bandaged firmly and evenly—firmly enough to prevent any internal extravasation. Lastly, external appliances suitable to the case should be put on. If, as in my first case, the superior fragment tends to ride forward, I use a double splint with transverse iron lips or brackets, and in all cases I should use extension.

My two cases were: (1) A boy, aged 13, operated on in the West London Hospital on March 11th, 1892; and (2) a man, aged 24, operated on there on December 20th, 1892. In the former case union of a fracture at the junction of the upper with the middle third of the femur had already taken place, and the bone had to be refractured. In the latter the fracture was ten days old. The amount of shortening, in spite of my house surgeon's efforts to abolish it, was in the case of the boy nearly  $2\frac{1}{2}$  inches clear, and in the case of the man 1 inch. The use of the pins absolutely prevented the slightest shortening. In the boy's case they were removed too early, namely, on the twentieth day, and he left the hospital with  $\frac{1}{2}$  inch of shortening due to angularity at the point of fracture. Absenting himself for a month, he then returned with more angularity still. In this case the pins were not kept in long enough, and he ought not to have been allowed to escape from observation. He slept well the night after the operation, and never complained of pain, nor had he any rise of temperature. The pins caused no irritation. They were merely improvised out of a stout old white-metal wire retractor (plated). The man lay in a long splint with 7 lb. weight extension, and the pins were *in situ* until the forty-third day after operation and the fifty-third after fracture. There was no pain and no sign of irritation, general or local. His temperature remained normal except two days after



operation, when it reached  $99.4^{\circ}$ . Firm union resulted without the slightest shortening. When the dressings were removed, the pins, which had been imbedded for forty-two days, were found to be a good deal rusted, exciting superficial suppuration at the orifices of the puncture, but no pain or rise of temperature. Under boracic dressings the punctures healed in a few days. I am sorry to say that this patient fell down owing to his crutch slipping, and rebroke the femur, but probably without tearing the periosteum, as there was no deformity, no shortening, and no swelling, but only tenderness and weakness.\*

It is demonstrated, therefore, that the pins can be left painlessly and apparently without doing harm in the femur, and that, in combination with simple extension and the long splint, they can completely prevent shortening. With regard to the best time for inserting them, I should recommend about ten days to a fortnight after the accident. By then effusion should have become either absorbed or organised, leaving only well-vitalised tissues to deal with any germ that might accidentally enter. By that time, also, it would be seen whether thorough reduction and ordinary appliances would suffice for the case or not, and at that period, also, the tracks made for the pins need not communicate with the fracture itself.

The PRESIDENT thought the method suggested was bold, ingenious, and likely to be beneficial in some cases, but for his own part he adhered firmly to the long splint, applied under anæsthesia with over-extension to 1 inch of lengthening. He did not approve of discarding the perineal band, as was done by some surgeons in modern times.

Mr. HARRISON CRIPPS remarked on the fragility of the pins shown. He thought the cases for which they might be applied were very few, and adverted to the danger of perforation by one fragment in the struggles resulting from the administration of an anæsthetic. The spasmodic contraction of muscles subsided by the third day, when the limb could be put up without any great effort.

Mr. OPENSHAW was in accord with the remarks already made by Mr. Cripps, and thought it was an advantage to have the edge of the splint bare and notched so as to exert extension throughout the limb by

---

\* In a fortnight the limb was apparently quite firm and strong again, without deformity and without shortening. Such a quick union will seem almost incredible to surgeons familiar only with ordinary fractures, which are, of course, caused by considerable violence and accompanied by great damage to the periosteum; but after refracture from slight causes reunion is usually very quick, probably because the fibrous parts clasping the fragments together are so little disturbed.

bandages rather than by the stirrup alone; by this means, without discomfort to the patient, sufficient traction could be exerted to overcome any degree of shortening.

Mr. KEETLEY, in reply, said he intended the method only for special cases. There was sufficient strength and rigidity in the pins, and the power required to keep the fragments in position was not great.

## THE PHYSIOLOGY OF DEATH FROM TRAUMATIC FEVER: A STUDY IN ABDOMINAL SURGERY.

By JOHN D. MALCOLM, M.B., C.M., F.R.C.S. Edin.

IN the clinical study of traumatic fever many facts may be observed which indicate that marked disturbance of the vascular system may be associated with a very slight rise of body temperature, and, conversely, that a decided elevation of the thermometric record may occur conjointly with no very evident alteration in the pulse rate. Recent investigations\* into the physiology of heat production, and of the increased temperature of the body during the febrile process, show that both heat production and heat elimination are under the control of the nervous system, and that each may occur independently of the condition of the vaso-motor system. Thus, both on clinical and experimental grounds, we are justified in considering the vascular conditions and the thermal conditions found during a traumatic fever as independent effects of one cause.

*The Local Vascular Conditions of an Inflamed Part.*—When an injury is inflicted on the tissues there is brought about a condition of the affected part which Lister has described as “bordering on loss of vitality, but quite distinct from it.”† The blood does not flow freely through the irritated area, and serum and leucocytes are exuded locally. The surrounding vessels are dilated, and through them there is a very free circulation. Other changes may take place, resulting in the formation of cicatricial tissue, but resolution may follow without any alteration of structure.‡

\* Macalister: “Gulstonian Lectures,” ‘Brit. Med. Jour.,’ vol. i, 1887, p. 670.

† “On the Early Stages of Inflammation:” ‘Phil. Trans.,’ vol. cxlviii.

‡ Watson Chyene: Heath’s ‘Dictionary of Surgery,’ Art. “Inflammation.”



Clinical observation shows that the evidences of local disturbance increase for about three days before signs of recovery begin.

*The General Changes in the Vascular System during an Inflammation.*—When these local changes are taking place there is a contraction of the vessels throughout the body,\* and this also, in normal conditions, reaches its maximum in about three days. It is usual for the pulse to become quicker and smaller at the same time that the temperature rises, but there is sometimes a marked divergence between the degree of the changes in the pulse and of those in the temperature. The heart action may be comparatively slow, and when this occurs the character of the pulse may be full and hard, or small and feeble. If the temperature be high after an operation, and the pulse at the wrist be small and feeble but slow, there is a bounding impulse in the large vessels, and there are loud, distinct heart sounds, with an accentuated second sound in the aortic and pulmonary areas. These conditions indicate distinctly that there is a rise of blood pressure, and that this is brought about by increased resistance in the peripheral vessels. It is obvious that the heart is beating powerfully, whilst the pulse is small and feeble. We must conclude, therefore, that although a small weak pulse may of course be due to debility of the heart, yet *feebleness of the pulse at the wrist is not necessarily an indication of cardiac weakness*. The small vessels may be so contracted that the blood-flow is almost excluded from them. The force and fulness of the pulse beats must be taken only as an index of the strength of the heart relatively to the obstruction induced by the contraction of the vessels between the artery examined and the heart. The heart action may be very strong, yet the pulse may be very feeble, or may even be absent in some of the vessels. Although the signs of this extreme contraction are only to be made out clinically in cases in which the heart continues to beat slowly, there can be no doubt that a similar contraction of the vessels takes place in all cases. If there be no complication the evidences of general disturbance of the vascular system disappear when the local abnormal changes are recovered from.

*The Relationship of the Local Vascular Changes to those of the Vascular System generally in Inflammation.*—A local increase of resistance to the blood-flow through an inflamed part would fully

\* Broadbent on Fever: Quain's 'Dictionary of Medicine.'



account for the contraction of the vessels throughout the body and for the rise of blood pressure which takes place.\* But there is an area of vascular paralysis around the inflamed centre which must tend to diminish rather than to increase the local intra-vascular pressure, so that a local increase of resistance to the blood-flow cannot be the cause of the general contraction of the vessels. It is obvious, however, that the effect produced on the nerves of the inflamed area—on the nerves of the tissues in which stasis exists—must result in an impression on the vaso-motor centre that blood is not flowing through the part; that although surrounded by an unusually active circulation there is in these tissues a want of fresh blood. The intensity of this impression on the nervous centre will increase with the extension of the local action. An attempt may be made on the part of the vaso-motor system to satisfy the necessity for an increased supply of blood to any particular area in two ways, namely, by local vascular relaxation or by a general contraction of the vessels throughout the rest of the body. Both these conditions are produced by an inflammation. The dilatation of the vessels surrounding an inflamed area was shown by Lister to be a local condition which may be produced in the limb of a frog after it has been separated from the body.† The object of this vascular relaxation may well be to admit more blood to the inflamed part. But, however great the calibre of the minute vessels may become, there still remains the central area of stasis and diminished blood supply. It is obvious that the continuous want of fresh blood in this area is an adequate cause of the general contraction of the vessels which occurs. It is, on the other hand, believed by many that the phenomena of traumatic fever are always due to the existence of morbid matter absorbed from a wound and circulating in the blood. Even in aseptic conditions it is said that a “hypothetical substance, ‘pyrogen,’ ” is produced from the damaged tissues, and is the cause of fever. In support of this view it is asserted that all the component parts of wound discharges are pyrogenous.‡ Without doubt, wound discharges and other substance, if injected into the blood stream, do act on the vascular system and on the heat-regulating mechanism. Certain substances, also, which are produced in putrefying

\* Landois and Stirling's 'Physiology,' 4th Ed., p. 141.

† 'Phil. Trans.,' vol. cxlviii, *loc. cit.*

‡ Victor Horsley: Heath's 'Dictionary of Surgery,' vol. ii, pp. 367, 368.

animal matter and in unhealthy sores, if introduced into the system, even in small quantity, either by injection or through a wound, will kill the patient with absolute certainty. This is not to be denied, but it is the object of this communication to show that the phenomena of fever, and now more particularly the vascular changes, may be explained without assuming the existence of any such poison, that, in fact, the changes which constitute the febrile condition are caused by peripheral irritations acting through the nerves on the nerve centres, a view which has already been ably advocated from another standpoint by Dr. Hale White.\* The conditions under which the individual constituents of healthy discharges may be separately introduced into the blood stream are widely different from those which exist in the case of a healthy wound. It seems to me that when fever is increasing, as the result of a healthy inflammation in a wound, vital action in the immediate neighbourhood of the injured part diminishes. The tissues become partially devitalised, whilst the blood and other fluids become stagnant. If the parts be kept absolutely quiet, resolution may take place with very little constitutional disturbance. But if local action be severe all the appearances which may be observed indicate that the exuded matters remain in the tissues of the inflamed part till resolution begins, while beyond the area in which this exudation occurs there is no evidence that the blood is altered directly by the local changes. If the irritation be sufficiently severe the central parts may actually die and be separated as a phlegmon. Thus, during the advance of a healthy inflammation it would seem that there is very little, if any, evidence that new products are thrown into the circulation from the action taking place in the injured parts. Absorption of serous matter must take place if union by first intention results when a wound is closed without due provision for drainage having been made. But the more quickly the absorption takes place the less is the disturbance of the pulse and temperature. It is when the discharges are not absorbed, when they collect between the lips of the wound or in the surrounding tissues and remain there, that febrile reaction runs high. With the spread of the local process there is a corresponding increase of the febrile phenomena. When

\* "The Heat Centre Theory from a Clinical Point of View:" 'Guy's Hospital Reports,' 1884. "The Theory of Pyrexia," 'American Journal of Medical Science,' Nov., 1890, and other papers.



inflammation is undergoing resolution the case is different. Then absorption of exuded solids and fluids must occur. There are probably unusual chemical reactions taking place locally, and resulting in abnormal chemical products, some of which must pass into the circulation. At this time, however, the contracted condition of the vessels and all the other indications of fever are subsiding.

*The Condition of the Vessels during Shock.*—A study of the phenomena to which the term “shock” has been given throws light on the question under consideration. It is very generally asserted that the condition of shock is due to a paralysis and dilatation of the vessels, especially of those governed by the splanchnic nerves,\* and it has been experimentally proved that these vessels may be so dilated as to produce symptoms like those of hæmorrhage.† There seems, however, to be reason for believing that in shock a contraction of the vessels occurs as in fever. If shock were due to a paralysis and loss of tone in the vessels it would be most unlikely that we should find the signs which may occasionally be observed in shock as in traumatic fever—namely, a small, feeble pulse with a strong, powerful, *slow* heart action. In one case I noted immediately after an operation an almost pulseless condition at the wrist, whilst the heart was beating 48 times to the minute, and manifestly with great force. The evidence to be derived from experimental inquiry is also in favour of the view that there is a contraction of the arteries in shock, for the stimulation of any sensory nerve seems to bring about a constriction of these vessels.‡

*The Effects of Exposure to Cold in Shock and in Fever.*—Exposure to cold after an injury greatly increases the severity of the symptoms of shock; indeed, a condition closely resembling that of shock may be induced by cold alone. The direct effect of cold is to cause a constriction of the exposed capillaries and a consequent increase of resistance to the blood flow through them. In health the influence of the vaso-motor system immediately comes

\* MacCormac: Quain's ‘Dictionary of Medicine,’ Art. “Shock.” Payne's ‘General Pathology,’ p. 153. H. C. Wood on Fever, p. 98. Victor Horsley: Heath's ‘Dictionary of Surgery,’ vol. ii, p. 434.

† Landois and Stirling's ‘Physiology,’ 4th Ed., p. 837.

‡ Lister: ‘Phil. Trans.,’ vol. cxlviii, p. 611. Landois and Stirling's ‘Physiology,’ 4th Ed., pp. 748 and 835.



into play, causing dilatation of the vessels leading to the parts where the capillary circulation is obstructed and also reflexly throughout the body. This is the healthy action caused by exposure to cold, and is obviously opposed to the condition of contraction of the vessels produced in fever. This action, therefore, seems to afford an explanation of the beneficial effect of cold applications in febrile conditions. In accordance with this view an immediate improvement of the pulse may often be noted when an ice-cap is applied for the reduction of fever. If, however, the temperature of the surface be lowered too much, the obstruction in the superficial vessels becomes so powerful that the compensatory dilatation of those leading to the surface is not sufficient to counteract the opposition to the bloodflow induced by the cold. The second action of the vaso-motor system then comes into play. The vessels elsewhere contract so as, if possible, to force the blood into the skin. The condition of shock may be thus produced, and this seems to be the explanation of the collapse which sometimes takes place when cases of high fever are treated by the cold bath. The internal parts of the body are the last to be affected by cold; hence in shock, unless death be instantaneous, the circulation goes on longer in the internal and warmer areas of the body. We therefore find an "enormous distension of the abdominal vessels."\* This, however, is not the cause, but a consequence, of the mode of death. It is to be particularly noted that in shock there is no time for the development of any poison; the symptoms must be due entirely to nervous action. It follows that the same effect may be of nervous origin in traumatic fever also, and it seems more rational to attribute the pulse phenomena to the action of a mechanism which is known to exist than to the presence of a hypothetical poison acting in a manner not known.

A general contraction of the vessels will, however, tend to set in action those nerves which indicate that a greater supply of blood is required by the various tissues. This must act as a check on the extent of the vascular constriction, and therefore the condition of the vessels during a traumatic fever depends on the comparative strength of the reflex stimulus to contraction arising in the inflamed area and of the normal regulating force of the vaso-motor system.

*The Effect of Constipation and Obstruction of the Bowels on the Condition of the Vessels in Traumatic Fever.*—It is well known that

\* Payne, *loc. cit.*

if constipation be permitted to continue after an operation an exacerbation of the fever is apt to take place. In abdominal surgery, serious disturbance, and even death, may be induced by retention of the contents of the bowels after an operation. The symptoms produced by this condition have been very generally mistaken for those of peritonitis. The diagnostic signs which distinguish inflammation of the peritoneum from a simple retention of flatus after an abdominal section had not, so far as I know, been differentiated until I published a paper on this subject in the autumn of 1887.\* About a month after my paper was read before the Medical and Chirurgical Society of London, Professor Olshausen made a communication to the Obstetrical and Gynecological Society of Berlin,† in which he advocated views to some extent similar to my own on this subject. He gave the cause of death in these cases the excellently descriptive name of “pseudo-ileus.” Six months later Dr. F. Verchère‡ published a paper in which he also described death from paralysis of the intestine following abdominal operations. Both these writers stated that this mode of death had not previously been described. Their papers seem to be attempts to define the same class of cases as those which I had described; but the conclusions which have been come to by Olshausen and Verchère differ widely and in most important respects from those which I have arrived at. This is a convenient opportunity for further discussing this question, as the conditions found in cases of pseudo-ileus throw light on the causes and also on the effects of contraction of the arteries in inflammation. During an abdominal section there is obviously a considerable likelihood that the various coils of intestine may be disarranged, and may be left in such positions that the downward passage of the contents of the gut will be hindered. The peristaltic action of the intestines usually unfolds any awkward turns which are thus produced in its lumen; but adhesions may take place very quickly, and may so absolutely fix the bowel in an unfavourable position that only considerable force will release it. The exposure and manipulation of the bowel also produce a temporary partial paralysis which favours the formation of adhe-

\* ‘On the Condition and Management of the Intestine after Abdominal Section,’ ‘Medical and Chirurgical Transactions,’ vol. lxxi.

† ‘Centralblatt für Gynecologie,’ January, 1888.

‡ ‘Revue de Chirurgie,’ July, 1888.



sions and diminishes the likelihood of the bowel being released by the force of peristalsis. When death takes place in these cases, evidences of peritonitis are usually found *post mortem*, but if the abdomen be reopened with a view to relieving an obstruction, evidence of peritonitis may be wanting, although the symptoms are well established, and sometimes after death there is no sign of this disease. Again, it may often be observed that when the symptoms are progressing they suddenly subside after an escape of flatus from the rectum.

When retention of the contents of the bowel occurs the condition of the patient is a most dangerous one. In a typical case of pseudo-ileus it is important to note that the signs and symptoms are exactly those of an uncomplicated case until an amount of fluid and gases sufficient to produce symptoms of obstruction has collected in the intestine. If there be no other complication, the initial symptom is usually distension of the abdomen, and this is first seen on the second or third day after operation, at or about the time of the highest temperature and pulse naturally arising from the traumatic fever. The thermal and vascular systems may show a tendency to assume their normal conditions whilst the distension increases, but if relief be not obtained there is always a return of the rise of the temperature and pulse. This may occur at a very variable period, from a few hours to two or three days before death. The pulse and temperature may rise together, but in these cases a most marked divergence between the extent of the changes in the pulse and in the temperature may be observed. The pulse-beat invariably becomes small and feeble as the abdominal distention becomes greater. The small pulse is due to contraction of the vessels of the body generally. The distension of the walls of the gut must lengthen, narrow, and compress its vessels and so cause an increased resistance to the flow of blood through them. This produces a reflex constriction of the arteries throughout the body and a general rise of blood pressure. As this occurs when the vascular tension is already high from traumatic fever, it is obvious that the effect on the pulse must be very great. Consequently in peritonitis with distension there is invariably found a very small thready pulse. That the small pulse in cases of pseudo-ileus following abdominal section is not necessarily due to cardiac debility is shown by a comparison of the pulse with the condition of the heart and large vessels, which in not a few cases



reveals the same relationship as exists in traumatic fever and in some cases of shock—namely, a small feeble pulse with a strong heart action.

*The effects of contraction of the arteries on the rate of the cardiac rhythm.*—The contraction of the arteries must throw a great increase of work on the heart, and it is clear that the cardiac muscle does not in ordinary circumstances work at its full power. It is also known that in many diseases of the heart and of the kidneys, when increased work is thrown on the former, cardiac hypertrophy occurs, and the heart's action is frequently slowed in accordance with Marey's law.\* If, however, the work of the heart be too greatly or too suddenly increased, a quick, feeble pulse results, or instantaneous death may occur.† It is a clinical fact that a weak heart is very apt to be a quickly beating heart. But too much work and weakness are terms entirely relative. Just as in active exercise, in which there is a strictly physiological but, it may be, an extreme condition, the rapidity of the pulse must be attributed to the extra work required of the heart, so the quickening of the pulse in fever and in shock is a simple result of the increase of work thrown upon the cardiac muscle by the contraction of the arteries. This matter is elucidated by a study of certain cases in which constriction of arteries and increase of blood pressure occur without any corresponding acceleration of pulse-rate. Such a case as the following indicates well the condition to which I wish to direct attention. The patient died of pseudo-ileus, but the pulse was beating 96 to the minute six hours before death. One hour before death the pulse could not be counted at the wrist, though the heart was beating loudly and distinctly 108 to the minute. The temperature in the axilla was 101.6° F. I have never seen any other patient dying in this way without the pulse rising for days before, and finally becoming uncountable at from 140 to 160 to the minute. At the *post-mortem* examination in this case it was found that the cardiac muscle was remarkably firm and healthy, and that the wall of the left ventricle was hypertrophied to quite twice its normal thickness, whilst its cavity was not enlarged and its valves were in every respect normal. The evidence of cases like this seems strongly in favour of the view

\* Foster's 'Physiology,' 3rd Ed., p. 178.

† 'Remarks on Failure of the Heart from Over-strain': Roy and Adami, 'Brit. Med. Jour.,' vol. ii, 1888, p. 1321.

that if the heart be sufficiently powerful it will not beat rapidly from the existence of a simple inflammation unless this be of the most intense character or accompanied by other conditions acting adversely on the heart. Consequently the quick pulse is due to weakness of the heart relatively to the amount of work required to be done by it. This applies not only to individuals but to physiological conditions. For instance, the undeveloped heart of the child is apt to beat more rapidly than that of an adult under similar circumstances. In the parturient woman also the pulse is characterised by a comparative slowness, sometimes in strong contrast to the state of the temperature. In women after delivery there exists an excess of cardiac power from hypertrophy of the left ventricle, and this hypertrophy is most marked in multiparæ,\* in whom also the slow cardiac action after delivery is most obvious.† It is open to conjecture that a similar hypertrophy may frequently occur in cases of abdominal tumour and chronic inflammatory disease, and may account for the truly marvellous recoveries that sometimes follow operations in such cases.‡ Although the character of the pulse depends on the state of the vessels more than on the strength of the heart, it may be said that cardiac weakness and cardiac overwork come to very much the same thing. There is, however, reason to believe that death from traumatic fever, from some cases of shock and from pseudo-ileus following laparotomy, usually begins elsewhere, and not at the heart. In cases of pseudo-ileus following an abdominal section, the evidence to this effect is most easily made out, because in these cases a contraction of the vessels is induced, whilst the inflammatory conditions are becoming less marked, and therefore the effects of contraction of the arteries are to some extent separated from the other results of the inflammation.

*Olshausen's and Verchère's explanation of the symptoms and of the cause of death in cases of pseudo-ileus.*—Olshausen and Verchère§

\* Playfair's 'Midwifery,' vol. i, p. 139.

† Otto Spiegelberg's 'Midwifery,' translated by J. Hurry, vol. i, p. 289.

‡ In a paper recently published by Roy and Adami on the 'Physiology and Pathology of the Mammalian Heart' ('Phil. Trans.,' vol. clxxxiii B, 1892, p. 270), it is shown by exact experimental methods that a rise of blood pressure in the systemic arteries causes an increase in the force of the heart's contractions, and that this increase of force may or may not "more than counter-balance the increase in resistance to the contraction of the left ventricle" caused by the rise of the blood pressure.

§ *Loc. cit., vide p. 194, supra.*



attribute the deaths in these cases to the absorption of some poisonous substance from the alimentary canal, and this is the explanation given by most recent writers of the effect of constipation on traumatic fever. This explanation is not altogether satisfactory. A simple obstruction of the bowels, as from a stricture, may be complete, and yet the patient may continue to exist for many days, even for weeks.\* Death is eventually due to a gradual exhaustion of the vital powers or to some accidental complication; but obstruction of the bowels during the first three or four days of convalescence from an abdominal section, if complete, is invariably fatal not later than three or four days after progressive symptoms of obstruction become well marked. The passage of flatus through the anus, a movement of the bowels, or the removal of a large quantity of fluid from the stomach by means of the œsophageal tube will alleviate the symptoms and may delay the result; but unless the obstructive condition be removed, death is inevitable. On the other hand, if the abdominal distension be permanently relieved, recovery may take place when the adverse symptoms are very far advanced. The phenomena induced are therefore clearly due to the combination of the effects of obstruction of the bowels with those of traumatic inflammation. Conversely, it may be observed that all cases of rapidly fatal obstruction of the bowels are complicated by some inflammatory mischief, injury, or strangulation of tissue. Verchère states that micro-organisms are found in the fluid in a hernial sac, especially if much manipulation has been employed in taxis, and that if such fluid be reduced into the peritoneum death results from the septicæmia which he has described. He argues that when intestinal paralysis occurs after a laparotomy, there is a peculiar condition of the walls of the intestine which permits a kind of filtration through the intestinal coats. Consequently there is an escape of intestinal fluids or putrid gases. These fluids and gases are charged with micro-organisms which find in the peritoneum a favourable nidus for their absorption, and septicæmia commences.† It is, however, to be noted that Verchère includes in his paper the consideration of a great variety of conditions—namely, accidental injuries to the abdomen and operations for herniæ of all kinds, as well as laparotomies [for various objects.

\* Treves on 'Intestinal Obstruction,' p. 302.

† 'Revue de Chirurgie,' July, 1888.



He joins together for the purposes of scientific evidence all these cases, whether the gut has been left without damage or has been seriously injured. To embrace the results of such a wide range of observation and to endeavour to reduce the pathology of so many varying conditions to one standard, tends rather to confuse than to elucidate the ideas which are formed of the pathological changes under consideration. A case of strangulated hernia, or of severe contusion or laceration of the bowel, should not be classed with a case of laparotomy, in which no injury whatever has been done to the intestine beyond its exposure and manipulation with the hands and with the sponges. Yet Verchère distinctly says the strongest contingent of deaths from paralysis of the bowels is found in the simpler cases. Verchère and Olshausen have separated cases of death due to pseudo-ileus from those due to peritonitis, but they have still confounded them alike in their clinical and pathological aspects. They have made up their clinical picture from a consideration of both simple and complex cases, but mainly from the former, their pathology being founded entirely on the conditions observed in complicated cases only. Verchère's statement that these cases show a well-marked facies abdominalis is certainly not correct. The patient may be quite sensible, cheerful, and hopeful till half-an-hour before death. Again, the temperature, according to Verchère, is normal or subnormal till the last moments, while the pulse is feeble and rapid throughout. This is not to be observed in an uncomplicated case of ileus, or pseudo-ileus, following a laparotomy. In making the vomiting a general symptom, as opposed to the local ones, Verchère evidently means to attribute this to the septic condition which he believes to exist. From the fluid which accumulates in the alimentary canal, it is assumed by Verchère, Olshausen, and many others, that a poisonous matter is absorbed, which is the cause of the symptoms—and of death, if death ensue. Nevertheless, it is easy to observe in these cases that whilst the obstructive condition continues large quantities of fluid are thrown into the alimentary canal, and are in great part ejected by the act of vomiting. During the continuance of this process the condition of the patient grows steadily worse. If, on the other hand, as not infrequently happens, the obstructive condition be suddenly removed, so that the flatus passes downwards, the vomiting at once ceases. It is evident that under these circumstances much of the liquid contents

of the bowel must be absorbed, for diarrhœa does not often occur; but the escape of flatus from the bowel and cessation of vomiting in these cases are invariably accompanied by a marked improvement of the general condition. There is often a sharp rise of temperature with this improvement, due to the tearing of adhesions. The pulse, however, rarely rises in proportion, and usually becomes slower and softer in contrast with the temperature change. Thus the patient becomes worse when getting rid of the fluid from the bowels freely, and improves when absorbing it. The evidence to be found in these conditions seems very inadequate to support the conclusion that a poison in the intestine is absorbed and causes the symptoms. It seems more likely that the vomiting is due to exactly the same causes as in simple obstruction of the bowels, and that the great quantity and character of the vomit may be explained in other ways.\*

*The Effect of a General Contraction of the Vessels on the Tissues.*—The evidence of the existence of any form of poisoning being thus defective, the question arises whether there is any other possible explanation of the signs and symptoms observed. Another, and I think a more rational, interpretation of the processes which precede death may be given. It has been seen that inflammation causes a diminution of the pressure in the small bloodvessels and increase of vascular tension in the heart and large vessels. This condition is aggravated by intestinal distension. It is obvious that if the contraction of the vessels becomes sufficiently intense in any particular area, either the affected area must be altogether deprived of blood or the regulating force of the vaso-motor system must come into play, and constrict some and, if need be, all the other vascular areas in an endeavour to force the blood into that first affected. Such an intense contraction of the vessels affects both the tissues and the blood itself.

First, as regards the tissues. In cases of obstruction following an abdominal section, the urine invariably diminishes in quantity and in specific gravity as distension progresses; but there is no albuminuria. The amount of urine in health depends on the difference of "pressure between the blood in the glomeruli and the pressure within the renal tubules."† Hence in diminished blood pressure within the glomeruli there is a sufficient explanation of the state of the urine in

\* *Vide* p. 205, *infra*.

† Landois and Stirling, 4th Ed., p. 522.



the cases under consideration. The pulse is always small when the urine diminishes in these cases. About the same time that this change in the urine becomes marked, or soon afterwards, the surface becomes very cold, evidently from deficient circulation in the superficial vessels, for the internal temperature may be rising to a great height at the same time. This may be observed during the last five or six hours or more of life. A condition of coma comes on with very slight warning shortly before death, the patient being usually quite sensible till the last half-hour or less. The exclusion of blood from the organs of thought and from the motor areas is a sufficient explanation of this change, and as we have seen that there is a gradually increasing constriction of the vessels, it is rational to assume that this is the cause of the coma also. The vital centres in the medulla are finally affected, and the lungs and heart cease to act. During the final moments of life, if the heart be listened to, doubt must in many cases at once arise as to the correctness of any explanation of the phenomena observed which assumes that cardiac failure is an important element in their causation. When the pulse is absolutely gone from the wrist, and the respiration is a mere spasmodic contraction of the diaphragm, the heart in some cases may be heard beating with wonderful regularity. There must be some vascular areas which are not, up to the very last, so constricted as to stop the circulation. Probably the circulation goes on longest in the central and warmer parts of the body. Hence, as in death from shock, there is often congestion of the internal viscera. The conditions observed point inevitably to the conclusion that in the cases under consideration the arterial system gradually contracts. If the cause continues and is sufficiently powerful, the blood is excluded from one area after another till the medulla oblongata is affected, and the patient dies. A gradually weakening heart would lead to the same results without any constriction of the vessels at all, but the effects of the constriction of the vessels are absolutely independent of the strength or weakness of the heart. Though the condition usually causes cardiac debility, yet the strongest heart cannot save a patient from death if an ileus or a pseudo-ileus supervenes on an abdominal section.

The advancing constriction of the vessels when sufficiently severe must affect the inflamed area in the same way as any other, for the dilatation of the vessels around this area is a local condition. But a



free supply of blood to inflamed tissues is essential to their satisfactory resolution. In cases of abdominal section, when distension of the intestine occurs as the primary changes in the wounded tissues are subsiding, the vascular supply to the healing parts must be shut off both by direct pressure and by reflex contraction of the vessels. As the tympanites increases, therefore, a return of inflammation, and that of an unhealthy type, is certain sooner or later to take place in the wound. It is not to be wondered at that when the fresh burden of a diffuse unhealthy inflammation is thrown on the system, death is inevitable and rapid. The secondary inflammation thus induced appears to be the cause of the rise of temperature which invariably occurs in these cases. In this lies the explanation of the difference of the effects produced by this condition on the pulse and on the temperature. The inflammation which causes the secondary fever is a consequence of the changes in the vascular system, and therefore the pulse changes precede the temperature changes.

In uncomplicated traumatic fever it is not so easy to differentiate the effects of the contraction of the vessels on the tissues; moreover, death from inflammation is very rare without the presence of some complication—most commonly septicæmia—which renders the symptoms less definite. With these cases of pseudo-ileus after laparotomy as a guide, it is, however, not difficult to detect in death from acute inflammation all the evidences of the vascular changes described. The contraction of the vessels also explains those cases in which death occurs from twelve to sixty hours after an operation, apparently from the severity of the procedure. Such cases are variously attributed to “shock” and “exhaustion.” The patients certainly rally to some extent from the immediate effects of the operation, so that the term “shock” is not quite appropriate; on the other hand, the term “exhaustion” should be reserved, as a rule, for cases of death after a much more prolonged illness. In the cases under consideration the contraction of the vessels due to the shock does not relax before that due to the inflammatory changes comes on with great severity. The urinary secretion is never free; it may be completely suppressed from the first. The patient does not become properly warmed; full mental activity is not restored. After a variable time death takes place, with all the evidences of intense contraction of the vessels. The fatal result depends on an excessive degree of injury as compared with the vital power of the patient and with the regulating power

of the vaso-motor system. Perhaps in these cases more than in any other the fatality may be attributed to uncomplicated traumatic fever. Such deaths only occur in very feeble individuals or after the most severe operations. They must be carefully separated from cases of acute septicæmia, which they may closely resemble, but which occur after any operations, and as readily in the healthy as in the feeble.

Here it may be remarked that the vascular conditions in death from inflammation, as above described, have a close similarity to those of death from old age. It has been said that "the great difference between death from old age and death from a sudden seizure is that in the former death commences at the periphery and terminates at the heart—the empire of death begins at the circumference and ends at the centre; whilst in the latter death commences at the heart and spreads over the body generally—death begins at the centre of vitality and gradually extends to its utmost bounds."\* If, however, the view of the pathology of death from inflammation described in this paper be the correct one, then death from traumatic fever is only a very rapid example of death beginning at the periphery and terminating at the centre. In respect of the vascular changes, death from inflammation is as much in contrast with death from syncope, due to cardiac weakness or disease, as is death from senility. In inflammation, as in old age, the heart is the last part of the body to give up the struggle. When the heart is so overworked as it is in asthenic fever, cardiac failure and sudden death may be easily brought about, and sometimes follow an imprudent or involuntary muscular effort. I have seen one patient die from sudden cardiac failure due to choking during an effort at deglutition. She was undoubtedly dying in the slow way which is common in cases of pseudo-ileus after a laparotomy, and nothing could have been more marked than the contrast between the usual mode of death in those cases and the sudden cessation of respiratory and muscular effort, the immediate and absolute extinction of all physical and mental power, which was brought about by a violent attempt to cough.

*The Effect of the Contraction of the Vessels on the Blood.*—The effect on the blood itself of the existence of the febrile condition is

\* Bichat: 'Recherches Physiologiques sur la Vie et la Mort,' Paris, 1805, p. 151 (quoted by Geo. W. Balfour in the 'Edinburgh Medical Journal,' March, 1887, p. 775).



great, and certain experimental and clinical facts are of importance as aids to our comprehension of the results produced. Constant interchanges go on between the fluids in the tissues and those in the bloodvessels, and also between the former and the contents of the lymphatics, whilst the lymphatic system also communicates directly with the venous system. When a shrinkage of the vascular capacity occurs, there must necessarily be a diminution of the amount of blood in the vessels, and the fluids of the body must therefore accumulate in increasing quantities in the tissues and lymphatics. Hence, when the arteries contract in inflammation, in the absence of venous congestion, there must be much tension induced within the tissues throughout the body. In a favourable case this tension is physiologically relieved by the action of the excretory glands, and especially of the sweat glands. It is to be noted that an extreme contraction of the arteries, or even the shutting off of the blood-supply altogether, will not prevent the action of these latter glands.\* Sweating indirectly removes much of the blood plasma. There is no evidence, however, that the blood-corpuscles are carried off in this way. If only the plasma were got rid of, a very great increase of the number of red blood corpuscles relatively to the liquor sanguinis would result. This, however, is not found to be the case. Dr. Lockhart Gibson has recorded that "the white corpuscles always rise in number and the red corpuscles always fall in number for a day or two after operations, and that without any regular proportion to the amount of blood lost."† The red blood corpuscles must therefore be eliminated in some way. In health these corpuscles are continually being broken up and are as steadily being formed; but the rate of formation and destruction varies. The changes are greater in youth than in old age, and are very active after food has been taken.‡ During the febrile process also there is evidence that the red blood corpuscles are destroyed in great numbers. The amount of this destruction at any time may be estimated by the amount of bile pigments formed.§ That there is not usually any very obvious sign of an excess of bile formation in an ordinary case of traumatic fever is no proof that such does not take place. "Normally a great part of the bile goes round in

\* Landois and Stirling, 4th Ed., p. 555.

† 'Journal of Anatomy and Physiology,' vol. xx, p. 133.

‡ Wm. Hunter, 'Brit. Med. Jour.,' Aug. 3rd, 1889, p. 239.

§ Wm. Hunter, *loc. cit.*



a circle from the liver into the duodenum, thence into the blood, so to the liver again, whilst another part is carried down by the contents of the intestine, and after becoming more or less altered passes out of the body with the fæces.”\* There are therefore no clinical means of measuring the bile formation, although bile sometimes passes in the stools in obviously excessive quantity during a fever. There can be no doubt, however, that an increased formation of bile, or of certain constituents of it, does occur during the febrile state. It has been shown that the formation of bile and of urea depends in large measure on the destruction of red blood corpuscles, and that these excretions are therefore necessarily increased or diminished together.† In fever the urea excretion is greatly increased, and the urine pigment derived from the hæmoglobin may be augmented twenty times.‡ There is therefore distinct evidence of increased destruction of blood corpuscles during the febrile process. Again, in cases of obstruction of the intestine or of pseudo-ileus after laparotomy, along with the increasing contraction of blood vessels, there is marked evidence of great destruction of blood corpuscles before the secondary rise of temperature takes place. There is very evident diminution in the amount of excretion by the kidneys, amounting to complete suppression before death; but there is invariably a great and increasing quantity of indican in the urine which may be observed before any signs of suppression are detected. The amount of pigment vomited is also very great, whilst after death the liver is dark coloured and the gall bladder is distended with black bile. This altered bile is certainly produced in very large quantity, and it must be concluded that there is a correspondingly great destruction of red blood corpuscles. It is further to be borne in mind that this excessive destruction of blood corpuscles is, in these cases of pseudo-ileus, contemporaneous with the contraction of the arteries and not with an increase of temperature. On the contrary, the temperature, sometimes at least, is falling when the vomiting of altered bile is becoming excessive. In shock nothing is known, so far as I am aware, of any effects of the condition on the amount of bile and urea excreted; but convalescence from shock is often preceded by bilious vomiting, which may be due to

\* Lauder Brunton: ‘Disorders of Digestion,’ p. 185.

† Noel Paton: ‘Journal of Anatomy and Physiology,’ vol. xx, p. 521.

‡ Landois and Stirling, 4th Ed., p. 413.

increased secretion of bile and other fluids. With the certainty that the blood plasma is diminished, there is thus a considerable amount of evidence that the red corpuscles are also destroyed and excreted in conditions in which great contraction of the arteries occurs. According to Dr. William Hunter, we should expect to find the plasma and corpuscles of the blood thus removed, if at all, in their normal proportions. He has shown that the most remarkable feature presented by the blood is the "power it possesses of maintaining a composition as rightly entitled to be termed stable as that of any other tissue of the body."\* During health the destruction of blood corpuscles is accompanied by a corresponding amount of blood formation, so that the blood remains nearly or quite constant in quality and quantity. Hence, increased formation of blood would seem to be a physiological consequence of excessive destruction of it. But, whilst the capacity of the vascular system remains small or is being reduced, increased blood formation can only lead to still greater blood destruction. The formation of such a complex entity as a blood corpuscle must, however, make great demands on the supplies of nutriment, and therefore any great increase in the activity of blood formation is quite sufficient to account for the extreme wasting of the fatty tissues and skeletal muscles which undoubtedly takes place during fever.

*The Temperature in Traumatic Fever.*—I now turn to the consideration of the temperature in fever. There cannot be any doubt that the contraction of the arteries to which I have endeavoured to trace so many of the symptoms which are observed in fever, may exist in a very marked degree without any elevation of temperature being found. Moreover, great disintegration of the blood may occur when the temperature is falling. It has also been pointed out by Burdon Sanderson that "the febrile augmentation of the urea discharge takes place immediately after subcutaneous injection of pus, *i.e.*, at a time which precedes the elevation of temperature."† It is therefore certain that an excessive destruction of the red corpuscles may take place when there is no elevation of temperature, and, consequently, that the elevation of temperature in traumatic fever does not depend on the increased destruction of blood corpuscles. In seeking for the cause of the elevation of temperature in fever, whilst attributing the excess of excretion

\* Arris and Gale Lectures, 1889: 'Brit. Med. Jour.,' vol. ii, 1889, p. 116.

† 'On the Process of Fever': 'Practitioner,' April, 1876.



of bile, urea, and the like products to the unusually active destruction of blood corpuscles necessitated by the advancing contraction of the arteries, it might be suggested that the rise of temperature is coincident with, and caused by, the compensatory increase of blood formation. But "the urea discharge remains\* excessive during the whole course of the fever." Therefore an excessive destruction of blood must go on also during the whole process. The patient, however, towards the end of an uncomplicated traumatic fever is evidently gaining strength and blood. At this time, not only must the blood be formed to replace that which is being destroyed, but, as the vessels relax, the increasing intravascular space must also be filled up. Hence increased blood formation must continue, if indeed it does not become more active, as resolution progresses. The temperature is, however, falling during this time, and therefore increased blood formation cannot be the cause of its elevation. Nevertheless, the formation of blood corpuscles in unusual numbers must involve a large amount of tissue change in many parts of the organism. Calorimetric experimental evidence shows that whilst these processes of excessive blood formation and blood destruction and the wasting of the tissues and their subsequent return to the normal are going on, there is an increased elimination of heat. The variations in the amount of heat production and heat elimination which take place in health are very great. The taking of food and starvation, active exercise and repose, the temperature of the surroundings, and other conditions have a marked effect on the amount of heat developed and eliminated. In health these conditions have little or no influence on the body temperature. Elevation of temperature, therefore, does not depend on increased production of heat. In fact, less heat is produced in fever on fever diet than in health on full diet.† It has been shown that an elevation of temperature may be brought about by a stimulation of a portion of the brain substance to the "mesial side of the corpus striatum near the nodus cursorius of Nothnagel."‡ The conclusion has been drawn that by the stimulation of a particular nerve area in the situation named "the thermogenetic function of the muscles is abnormally

\* Burdon Sanderson, *loc. cit.*, quoting Senator: 'Untersuchungen über den Fieberhaften Process und seine Behandlung.'

† Wood on Fever, p. 239.

‡ Macalister: 'Gulstonian Lecture,' 'Brit. Med. Jour.,' vol. i, 1887, p. 670.



increased.”\* But, if an elevation of body temperature does not depend on an increased production of heat, then no such simple explanation of the thermogenetic, or rather heat-raising, effect thus produced is satisfactory. A more rational view of the thermal phenomena of fever is that elevation of temperature depends in no way on increased production of heat, but on something which disturbs the arrangement whereby heat production is counterbalanced by heat elimination. This undoubtedly sometimes occurs, and there can be little doubt that the mere stimulation of a thermogenetic tract will not cause a rise of body temperature so long as the rest of the heat-regulating mechanism remains intact. With this mechanism in a healthy condition, an extra development of heat would at once produce compensating thermolysis, as when food is taken and after violent exercise; therefore thermolysis must be interfered with at the same time that thermogenesis is stimulated, or the temperature will not rise.

Every assumption that the muscles are in a special manner the heat-producing tissues, that they have a thermogenetic function peculiar to themselves, and that on the excessive exercise of this function the febrile condition depends, seems to me open to objection. Doubtless the changes which take place in fever, and which lead to marked wasting of the muscles, are associated with the development of heat in the muscles, and are not dependent on, nor associated with, the exercise of the contractile function of their tissue. I have, however, shown that there is some reason to suppose that the wasting of the muscular and fatty tissues during the febrile process may be secondary to the efforts made by the organism to supply fresh blood in place of that which is destroyed. It might be argued from this that the blood-forming organs have the greatest claim to be described as specially fever-producing. We have, however, seen that the blood-forming function is also very active when fever is abating. A special claim to the title of thermogenetic cannot be established for any tissue. Probably all the tissues are thermogenetic in proportion to their bulk and vascularity. An explanation, which I believe to be new, of the rise of temperature in traumatic fever has suggested itself to me. It has been shown, as already pointed out in this paper, that whilst the

\* *Loc. cit.*

inflammatory process exists there is in the affected part a temporary partial devitalisation of tissue which is the primary lesion of inflammatory congestion.\* There is also evidence that during the advance of an inflammation an increasing area is subjected to this condition of diminished vital activity. I have endeavoured to show that this local condition, although the parts are full of more or less stagnant blood, and are surrounded by a very active circulation, may transmit to the vaso-motor centre an indication that there is a want of fresh blood in the part. Exactly in the same way, it seems to me that, although the parts are surrounded by an active circulation of blood which is above the normal body temperature, and though they are themselves actually above that temperature, yet, in the process of dying or becoming partially devitalised, as the normal local chemical reactions cease, the nerves of the affected tissues must transmit to the central nervous system an indication of increasing physiological inactivity—of the approach of the coldness of death. This would be expected reflexly to stimulate heat production. But if the body temperature be raised those tissues which eliminate heat—notably the skin—will be excited to increased action. Thus the local condition tends to raise the temperature whilst the heat-regulating mechanism endeavours to bring it down to the normal, and on the comparative power of these opposing forces depends the result at any particular moment. It is to be noted that, though I have attributed the changes in the pulse and in the temperature to separate physiological mechanisms, they are traced to the same cause—the partial devitalisation of the inflamed tissue; hence, in the condition of simple fever the pulse and temperature rise and fall together, but this correlation may be altered by various complications.

*The Specific Fevers.*—The foregoing conclusions apply to the simplest forms of fever only. It would appear, however, that in every fever all the conditions which I have described may be detected. In the specific fevers and in the septic fevers there is evidence that some processes are taking place besides and beyond those which constitute a simple fever arising from an injury. The superadded causal condition is a specific poison giving rise to the pathognomonic signs of the particular disease produced. Of the fevers thus brought about the septic varieties are especially deserving the attention of the surgeon. By septic fever and by

\* Lister: 'Phil. Trans,' vol. cxlviii, p. 698.



septicæmia I mean a complication of traumatic fever due to the absorption of a poison through a wound. The admission of a poison through a mucous membrane, as described by Olshausen and Verchère, has been regarded by some as a mode of development of septicæmia. I have endeavoured to show that in the case instanced death is due to other causes. Cases may occur, however, in which a poison which requires no wound for its entrance gains access to the tissues of a patient who has been operated on. It is believed, for instance, that operation cases and recently delivered women are specially susceptible to the poison of scarlet fever. All such cases should, if possible, be clearly separated from those of surgical septicæmia. Such an essential difference in etiology forbids that they should be classed together. The clinical symptoms and *post-mortem* signs of a septicæmia are those of a universal irritation added to those of a traumatic fever. Except in cases of septic intoxication, micro-organisms have been found in all the tissues of the body, but of all the organs the kidneys are most constantly affected in this way,\* and clinical evidence is very strong that through the renal tissue the elimination of the poison is mainly effected. In all forms of the disease if, and so long as, the kidneys continue to act there is a possibility, even a probability, that the patient may recover; but if albuminuria and suppression of urine occur, and are at all continuous, death will speedily follow. We cannot, however, attribute death from septicæmia with suppression of urine after an operation to retention of physiological effete matter which ought to be excreted. As I pointed out in regard to obstruction of the bowels after an operation, so in this case, death is too rapid to be due to the complication alone. Septicæmic suppression of urine rarely lasts more than three days, whereas in simple obstructive suppression of urine life is usually prolonged for from nine to eleven days.† Septicæmic suppression of urine leads, however, to great interference with the circulation of the blood through the kidneys, and thence arises a physiological condition in every respect parallel to that which exists when the blood-flow through the mesenteric vessels is interfered with by intestinal distension in cases of obstruction or paralysis of the bowel after laparotomy. Death may be induced in exactly the same way in both these conditions. There are, therefore, many

\* 'Pathological Transactions,' vol. xxx, p. 13.

† Sir Wm. Roberts: 'Urinary and Renal Diseases,' p. 27.



resemblances between the two classes of cases, but the only common cause of the exacerbation of the inflammatory fever in septicæmia and in pseudo-ileus is the obstruction to the flow of blood through a large and important vascular area. So like, however, are the two modes of death that Olshausen and Verchère, though recognising that there is a difference, have attributed the symptoms of pseudo-ileus to a species of septicæmia peculiar to itself. It may be stated generally that any complication which raises the blood pressure during a traumatic fever is dangerous, and, if persistent, may induce death in the manner which I have described.

Those who do not attribute the changes caused by even the simplest fever to peripheral irritation, assert that the phenomenon observed are always due to the formation of some poisonous substance, which, circulating in the system, induces the febrile condition. If it be assumed that peripheral irritation is not the cause of fever, both the hypothetical substance which produces this disturbance and its mode of action have yet to be discovered. On the other hand, the views of traumatic fever which I have endeavoured to formulate, explain also how certain irritant poisons may cause all the characteristic phenomena of the febrile state. Circulating in the system, these poisons must give rise to a widespread irritation and consequent partial devitalisation of tissue. Such a widespread partial devitalisation of tissue must produce the same effects on the vascular and thermal systems as are brought about by the more obvious devitalisation due to a local irritation. The effect may be slight or so severe as rapidly to induce great contraction of the vessels, a high temperature and death. It is even conceivable that death may be produced almost with the suddenness of shock. In accordance with this we know that in scarlet fever, for example, a fatal result is sometimes brought about before any pathognomonic signs of the disease show themselves. Cholera and certain snake poisons may also kill with extreme rapidity. The peculiarities of the poison, of the individual, and of the tissues, fully account for the conditions found in the special diseases produced. In every febrile disease there is evidence of peripheral mischief. Sapræmia, septicæmia, pyæmia, and all the exanthemata show signs of a widespread irritation with a tendency to inflammation in certain tissues and organs.\*

\* The Editors regret that, owing to a misunderstanding, this paper appears in a more condensed form than the author originally intended.

Dr. HALE WHITE thought there was no doubt that in some cases traumatic fever was the direct result of peripheral irritation, and mentioned the rise of temperature associated with the passage of a gall-stone or the existence of constipation. He referred also to the rise of temperature in meningeal hæmorrhage, in the status epilepticus, and hysterical pyrexia, in all of which the absence of bacteriological influence seemed certain.

The PRESIDENT questioned if, in the abdominal cases alluded to, the absence of peritonitis could be affirmed.

Mr. MALCOLM, in reply, mentioned several cases in which no evidence of peritonitis was discovered after death, although the clinical symptoms could not be distinguished from those usually attributed to peritonitis. Referring to some remarks by Mr. Alban Doran, he said that, in considering this subject, he excluded all cases in which there was an obvious source of septicæmia.

---

*February 20th, 1893.*

## ON SOME CLINICAL VARIETIES OF CHRONIC ALBUMINURIA, CHIEFLY WITH REGARD TO PROGNOSIS.

By C. H. RALFE, M.A., M.D. Cantab.

THE clinical significance of albuminuria has, undoubtedly, considerably diminished in importance during the last twenty years. Referring to text-books published about that time, one finds hardly any mention of the fact that albumen might appear in the urine independently of disease of the kidneys; or, if spoken of, it was recorded as "highly exceptional" and "unimportant." Moreover, in consequence of the fatality attendant on recognised inflammatory diseases of the kidneys, the presence of albumen in the urine came to be regarded as a symptom of the gravest import. Since, however, the practice of systematically examining the urines of all patients for albumen, as well as those of a presumably healthy class that present themselves for life assurance and for the public services, came into vogue, a numerous and well-defined group of cases has been determined, which for convenience may be designated as "functional," and which perhaps constitutes from one-third to one-half of all the cases of albuminuria that come under observation. Besides which, with regard to the albuminuria asso-



ciated with actual disease of the kidneys, the earlier recognition of the symptom, owing to the systematic examination of the urine—now, it is to be hoped, universally employed—points to the mischief in the primary stage of its inception before the structure of the kidney is irreparably damaged; and permits, by improved dietetic and hygienic measures, of checking its advance, and possibly in acute forms its recurrence. This has made the prognosis of albuminuria not only more hopeful, taken as a symptom generally, but even in the graver cases, when associated with renal disease, less immediately fatal than was, till quite recently, the case. It is in respect to this latter class of albuminuric subjects that I wish to invite discussion, and I venture to express the opinion that, when early recognised, and when the patient has been placed under proper supervision—dietetic, hygienic, and therapeutic—those forms of chronic albuminuria collectively known by the term “Bright’s disease” have a less dark prognostic future before them, but are even, for a time, susceptible of a very distinct amelioration. But in these cases (organic renal disease) there are such clinical varieties depending etiologically on the constitution of the individual—heredity; specific conditions, such as gout, syphilis, struma, &c., either acquired or inherited; specific poisons, such as scarlet fever, diphtheria, lead poisoning and the like—which have to be taken into consideration in coming to a conclusion as to the course any given case has to run, that nearly every case presents itself as showing some variation worthy of notice, either as affecting the predisposing or exciting causes or influences, or conditions that accelerate or retard the advance of the disease. I now propose to submit a few of the most important of these clinical variations and their influence on the prognosis of the disease.

The most familiar varieties of chronic albuminuria which present themselves are those which eventuate in the small red and small white granular kidney. Though so frequently coming under observation, still, owing to the insidious nature of their onset, they are rarely observed in their earlier stages, and there is, therefore, comparatively little information regarding their evolution, and the possibilities of arresting or retarding their development. When they do come under observation, the disease is usually far advanced, some decidedly unfavourable symptom having drawn attention to it. Taking the cases associated with small red kidney first into consideration, and excluding hospital patients in whom



the systematic examination of the urine when first coming under observation draws attention at once to the condition of the kidneys (and the same may be said of those presenting themselves for life assurance), attention is usually drawn to the true character of the disease by some symptom which denotes how far it has already advanced—as, for instance, attacks of asthma (renal dyspnœa), failure of sight (albuminuric retinitis), hæmorrhages from the mucous surfaces, and paralysis, all symptoms of most grave prognostic import, denoting advanced arterial degeneration and deterioration of the blood, after the recognition of which hopes of prolonging life are seriously curtailed, and, as a consequence, the prognosis of this form of renal disease is deeply gloomy. One question, therefore, I would submit is: Supposing the disease is detected in quite an early stage of its inception, is there any possibility of retarding its progress and rendering the prognosis somewhat brighter? As I have said, one so rarely meets with a case whose whole history is known, that such a question can only be answered by the collective experience of many observers. Personally I have observed only one case, and even that not quite completely, for, happily, the patient is still living. In this instance I originally saw the patient for an attack of bronchitis in 1878, when he was 55 years of age. A note was made at that time to the effect that there was no marked emphysema of the lungs, no enlargement of the heart, and no displacement of the apex. The pulse was 88, full and compressible. The urine was normal in general character, its specific gravity 1025, and depositing urates; there was no albumen or sugar detected. This patient again came under observation in 1882, complaining of headache, insomnia, and palpitation of the heart. Physical examination showed hypertrophy of the left ventricle and displacement of the apex towards the nipple line; the pulse exhibited some degree of tension. The urine was abundant, its specific gravity 1014, and it exhibited a trace of albumen. The patient was placed on a non-stimulating diet, chiefly milk, fish, farinaceous food, and white meats, whilst red meats, wine, and coffee were forbidden. He went to Homburg that summer, with the result, for a time, that the albumen disappeared. He has now been under observation for eleven years, with the result that, with the exception of a slight outward displacement of the apex of the heart to outside the nipple line, of a more palpable thickening of the radial vessels, of the

more constant passage of albumen, and of a urine of low specific gravity, he has not altered much for the worse during that period. He has adhered steadily to the diet prescribed for him from the first, and certainly up to the present time has not developed any serious symptoms of the disease, though, of course, it is impossible to say how long his vessels may resist the tension in them, or how far they have escaped degenerative changes, and his life may yet be prolonged. Still, such a case is instructive, in that, under moderate dietetic restrictions, the disease has not made further advances during the eleven years it has been recognised. When one reflects that this form of chronic renal disease is markedly hereditary it is possible, by keeping a watch for any manifestations of renal disease or antecedent cardio-vascular trouble and by placing the patient under proper dietetic control, to greatly hinder the advent of those symptoms which one is unable to combat successfully when once they have made their appearance.

With regard to the form of chronic albuminuria associated with the pale or mottled granular kidney, owing probably to the symptoms being detected earlier, the immediate prognosis is not so grave as in the preceding instance. Here the prognosis of any given case depends greatly on the nature of the predisposing and exciting conditions. Thus in the chronic nephritis of gouty patients the intensity and continuance of the inflammation are influenced by the frequency and severity of the gouty manifestations. These cases run often quite a chronic course, and when patients can avail themselves of the dietetic and hygienic regulations now advocated they have been known to live many years. Thus a patient who was seen by the late Dr. Murchison as long since as 1874, for nephritis associated with gout, is still living. He has wintered ever since in some dry warm climate, and has adhered to a non-stimulating and comparatively non-nitrogenous diet, and though the albuminuria has persisted throughout, he enjoys a fair measure of health, and as yet has developed no symptoms of uræmia or dropsy. Even when these symptoms manifest themselves, they are not of so grave import as their occurrence in some other forms of chronic albuminuria would lead one to expect, and the patients often experience relief from them on the outbreak of a frank attack of gout. In one case, a patient aged 40 years, who for three years previously had suffered from albuminuria,



was seized with deep coma preceded by convulsions, whilst he also had considerable œdema and almost complete suppression of urine. The case was regarded as almost hopeless, but after many hours the coma passed off, and shortly afterwards an attack of frank gout developed. He lived five years, continuing his work nearly the whole time, and had he taken care of himself, and his occupation been a less trying one, he would probably have lived even longer. In another case, a lady, the duration of life was seven years after the first attack of uræmia. In the nephritis occurring in patients who have suffered from syphilis or malaria, and especially those subject to lead poisoning, the prognosis is far less favourable, probably from the fact that degeneration of the vascular system occurs earlier. Indeed, it may be taken as a general statement that patients rarely survive more than from eighteen months to two years after the appearance of serious hæmorrhage, either from mucous surfaces or from the vessels of the retina. Mentioning the occurrence of hæmorrhage from degenerated vessels brings to my notice a comparatively rare form of nephritis, which is attended throughout with a hæmaturia more or less persistent. Of three cases recently under observation at the London Hospital, in two the hæmaturia was moderate but constant, in the third profuse. The first case had suffered from albuminuria for about a year, during which time the urine had been bloody; the amount of blood was never excessive, being mostly observed when deposited as a fine red line on standing. The albumen was not excessive, and after the patient had been kept at rest in bed and on a milk diet it became merely a trace. Still blood corpuscles were always to be found on microscopic examination, and on frequent occasions crystals of free uric acid. He suffered at the same time from eczema of the scalp. His general health was not impaired, and whatever the cause of the hæmorrhage it did not apparently proceed from vascular degeneration. The second case was that of a young woman, aged 23, who was admitted to the London Hospital suffering from hæmaturia. This at first was supposed to be due to possible renal calculus, but a close observation negatived that supposition, and there was no doubt that she was suffering from chronic nephritis. She was nearly three months in hospital, and during the whole time very definite traces of blood were always found in the urine, whilst the amount of albumen was never great. In this case the deposit



of uric acid crystals in the urine was frequent, and her father was stated to have suffered from gout. In the third case, that of a man aged 38, who was under the care of a colleague, the hæmorrhage was profuse. The patient had suffered from acute attacks of gout since the age of 11, and for some years past had suffered from hæmaturia, alternating with the gouty paroxysm. In this case, as in the others, there was no evidence of vascular degeneration, nor was the patient's health impaired to the extent usually found in chronic nephritis, and the disease, whatever its nature, was apparently running a comparatively mild course. The albuminuria became merely a trace whilst the patients were kept on a milk diet and at rest, and had it not been for the continued presence of blood in the urine little importance would have been attached to their condition. In another case of hæmorrhagic nephritis somewhat similar conditions were observed, only the attacks were paroxysmal, so that had it not been for the presence of actual blood in the urine it might have been taken for hæmoglobinuria. The patient's (himself a medical man) account of his attacks was briefly as follows: On the first occasion he had been feeling distinctly unwell three or four days before, with a vague feeling of malaise, biliousness, noises in the head, and intense languor. Then came a distinct rigor; the urine became scanty and smoky in colour, exhibiting under the microscope numerous blood corpuscles mixed with numerous uric acid and oxalate of lime crystals. This state of things would continue for some days and then the attack passed off, though blood corpuscles would readily reappear in the urine on any over-fatigue or indiscretion of diet. The quantity of albumen observed at any time was always comparatively a mere trace, and that not always observable in each sample of urine passed in the twenty-four hours. Before I saw him he had three such attacks, and I saw him just after his last. His urine contained only a trace of albumen, but many blood corpuscles and crystals of uric acid and of oxalate of lime. I should state that during the paroxysms his pulse, which was usually soft, became hard and tense. A case somewhat like this, only the hæmorrhage was more profuse, I have seen from time to time during the last nine years. He was originally sent as supposed to be suffering from renal calculus, as he complained frequently of paroxysms resembling renal colic and profuse hæmaturia. That his trouble was not caused by stone was soon proved, since it

was evident the paroxysm did not follow exertion; and also, as soon as the hæmaturia cleared up, albuminuria persisted in considerable amount, whilst the urine contained casts of recent and old origin. In this patient the paroxysms were preceded by the same feelings of malaise as in the other patient. Both cases improved greatly when placed on non-stimulating diet and both had a prolonged rest, the first passing a winter in Egypt, the other living for some time in a mild and sheltered spot in Ireland. The first patient reports that since his return he has rarely experienced a paroxysm, and that only at long intervals and in very slight degree. The other states that he has had no paroxysmal attack since he has developed gout freely in his knees, wrists, and smaller joints. It is interesting, in considering the nature of this hæmorrhagic nephritis, to bear in mind that the nephritis occurring in malarious subjects is especially distinguished by its hæmorrhagic character, caused, no doubt, by the intense congestion of the abdominal viscera. May one, therefore, not conjecture that the gouty poison may have a similar effect in cases where the hæmaturia is distinctly of a paroxysmal character, and that the kidney itself is the seat of a true gouty inflammation?

So far I have been considering chronic albuminuria as the result of a hyperæmia more or less active, and must now consider the albuminuria resulting from passive congestion. Of late years this has not received the attention it deserves, partly because attention has been more fixed on those chronic forms of nephritis associated with cardio-vascular changes and increased arterial tension, and partly because such cases are less numerous in hospital practice than amongst private patients. In its most complete form this albuminuria is met with as a result of valvular disease of the heart, causing what is termed "cyanotic induration of the kidney." So far as prognosis is concerned in cardiac disease, the appearance of albumen in the urine denotes nothing more than the increasing failure of the right side of the heart and stasis in the renal veins. But there are also other cases in which venous stasis undoubtedly plays a part in causing a chronic albuminuria independently of actual disease of the heart. These occur chiefly in middle-aged persons who, either from hereditary predisposition or too free indulgence in rich food and generous wines, have become obese, especially as regards the deposition of fat round the abdominal viscera. Such individuals are of a lax constitution and usually



have a feeble circulation, as a consequence of which assimilation is very imperfectly performed, and they require to be well supported in order that enough material may be assimilated by their imperfect organs to carry on the vital processes. These patients, owing to the laxity and feebleness of the muscular system, take little exercise, with the result that they become flabby and, especially the abdominal, muscles relaxed, so that the abdominal viscera are not supported and the whole abdomen tends to fall forwards. This condition, together with the little exercise taken and the amount of food ingested, adds to the already existing abdominal plethora, and after a time these patients, who have already sought advice for relief from some dyspeptic trouble, begin to pass albumen with their urine. At no time is this ever excessive, and can be easily kept in control by diet and hygienic measures, and especially by the use of mild aperient waters. In none of the cases I have had under my observation have I noticed the development of active gout or hypertrophy of the heart or degeneration of the vessels, nor are uræmic symptoms usually present. When these patients die—and they often attain old age—it is from an intercurrent attack of some acute disease. So long, however, as these patients are not exposed to any vicissitude, are well fed—for in this form of albuminuria a more liberal dietary is required—and are warmly clad, they live for many years without any apparent advance of their renal trouble. This form of chronic albuminuria is often associated with an equally mild form of diabetes, both running a course together for many years. In five cases in which I have watched the progress of this double disorder, in none did it seem to affect greatly the general health. All except one who is still living, and he is 65 years of age, lived till close on their 70th year, whilst the average duration of this disease in these four cases was just over twelve years. It is not improbable that the albuminuria met with in morphia *habitués* is of this congestive character; at all events, I feel disposed to accept the views of Professor Huchard, of Paris, on this point, although the arguments on the other side as expressed by Dr. Haig are deserving of consideration.

The albuminuria associated with lardaceous degeneration of the kidneys usually runs a protracted course; so much so, that one often loses sight of the case before its termination. In four cases, however, which have come under observation whilst preparing materials for this paper, one, a medical man, had suffered from



enlarged liver and spleen with albuminous urine for twelve years; the second, a medical officer in the army, had been invalided from the service fourteen years previously on account of the disease; another case was that of a woman in whom the disease seemed to have followed repeated and severe attacks of ague, contracted twenty years before when residing abroad; in a fourth case, also a woman, the albuminoid degeneration was complicated with visceral syphilis, but her illness dated back many years. In fact, the chronic albuminuria of lardaceous degeneration runs, as a rule, a protracted course unless it becomes complicated with nephritis, when the downward course is extremely rapid. This unfortunately was brought under my notice last year in the case of a member of our profession. He had suffered from albuminoid disease for twelve years till, when away for a holiday, he received a chill, and suffered subsequently from hæmaturia. From this time the character of his urine changed—it became less profuse and contained casts, which had not been detected before. Up to this time he was engaged in the active duties of his profession, and felt little the worse for his chronic complaint; now he became frequently indisposed; the heart, which up to that period presented nothing abnormal, now enlarged rapidly, and extensive arterial degeneration soon became evident. He usually experienced an attack of gout in the spring of each year, but none developed this year, and he died in the summer from an attack of cerebral hæmorrhage.

Lastly, with regard to the forms of albuminuria not associated with renal disease; they have but slight prognostic significance, and are mostly transient and intermittent. This is so, no doubt, during the earlier years of life, up to 30 or perhaps 40; but the occurrence, especially for the first time in middle adult life, of an intermittent albuminuria is often a danger signal indicating more serious mischief to follow. There may be no evidence of changes taking place in the kidneys, no enlargement of the heart; the albumen disappearing, especially after a course of Homburg or Carlsbad waters, but reappearing on the slightest provocation, till eventually undoubted evidence of contracted kidney is present. A physician consulted me in 1880 for an intermittent form of albuminuria that seemed to follow upon slight dyspeptic troubles; and which passed away when he paid attention to the bowels. His heart and vascular system were perfectly normal. During his sojourn in Europe he had consulted many physicians, who all

encouraged him by speaking of it as "a functional albuminuria." He was then a hale, hearty man of about 50, but within four years of that time the heart became enlarged, the albuminuria persistent, and his vessels degenerated. Had, I think, more attention been paid to this premonitory albuminuria and a rigid non-stimulating diet insisted on, the onset of more serious symptoms might have been delayed.

Having thus briefly and very inadequately mentioned some of the chief varieties of chronic albuminuria, I hope I have indicated that the two chief factors which have an effect in brightening prognosis and adding to the duration of life in these affections are, firstly, the earlier stage at which the disease is detected owing to systematic examination of the urine of all patients who seek advice; and, secondly, the improved system of diet now insisted on. The profession is much indebted to Sir George Johnson's teaching and his advocacy of a non-stimulating diet in inflammatory affections of the kidney. This of late years has taken the form of the "milk-cure," feeding the patient absolutely on milk; or if not entirely possible, with the addition only of the slightest modicum of other food, and that of a non-stimulating character. This so-called "milk diet" is now so generally adopted that it might be considered superfluous for me to draw attention to it in this paper. But, in spite of its general adoption, there have always been some doubts expressed as to whether its administration is equally useful in all cases, and even if it may not be deleterious in some. With a view to obtaining information on this point, I have instituted for some time past a series of observations on the effect of milk diet on the different forms of renal albuminuria. These observations consisted in making an analysis of the patients' urine on admission into the hospital, and afterwards gradually putting them on a milk diet (from 3 to 4 pints a day). This was continued for four weeks, a weekly analysis of the urine being made with regard to the amount of urine passed and the amount of urea, albumen, and total solids. At the end of these four weeks a mixed diet was gradually resumed and another analysis made. As a result, in five cases of acute or subacute nephritis with dropsy an increase of urinary water, of urea, and a diminution of albumen were observed, which when the ordinary diet was resumed showed a tendency to relapse. In two cases of albuminuria associated with cardiac complication a slighter im-



provement was manifested. With one well-marked case of albuminoid degeneration the milk diet was distinctly unfavourable, so that after a fortnight's trial it had to be discontinued and a more generous dietary adopted, with distinct benefit to the patient. In three well-marked instances of contracted kidney with marked vascular degeneration the milk diet was not at all well borne by the patients. In one case, on the second day of its trial the patient was seized with severe uræmic convulsions; and in the other two aggravation of already existing uræmic troubles became more pronounced on a persistence of an enforced milk diet; whilst in all three an improved condition was noticed when a more solid dietary was resumed.

From these observations amongst hospital patients and from the experience obtained from private patients, I have come to the conclusion that the best results are obtained from the milk diet in cases of acute or subacute nephritis in which the diuretic action of the lactose in the milk increases the flow of urine and relieves the dropsy; whilst in cases where degenerative changes have taken place, with an already failing heart, it is better to give a more solid and more stimulating food than milk, care being taken at the same time that it is easily assimilable and not highly nitrogenous. To my own mind the employment of an exclusive milk diet in chronic nephritis is positively injurious after degenerative changes have manifested themselves, yet in the earlier or more acute stages of the disease it must form the basis of treatment, and if it cannot be persisted in should be employed either in a modified form or resorted to whenever an exacerbation occurs.

Dr. STEPHEN MACKENZIE considered that in the paper just read by Dr. Ralfe he had added considerably to our knowledge, and that by leaving the somewhat beaten paths he had drawn attention to some types of chronic albuminuria not generally recognised. In the first place he agreed with Dr. Ralfe that the prognosis in granular and gouty kidney was not so serious as used to be imagined; or, rather, that since the time of Bright the increase of our knowledge made known to us that patients with contracted kidneys might live for many years. He mentioned several cases in which he had watched patients for eleven or twelve years. Like Dr. Ralfe, he thought that dieting was of the greatest value in such cases, and one of his patients had taken no meat food for many years. A second point of interest in Dr. Ralfe's paper was his remarks on hæmaturia in connection with granular kidneys. He had not watched any cases over such a protracted period as those related by Dr. Ralfe, but he had seen some cases, and Mr. Busby had drawn attention to them. In a case he had seen lately, cancer of some part of the urinary tract



was suspected, but he found casts and albumen in excess of that accounted for by the presence of blood, and concluded it was a case of granular kidney, as was proved by *post-mortem* examination. He thought Dr. Ralfe had distinctly described a type of cases of chronic albuminuria not usually recognised, but which his own experience confirmed—persons of stout habit, flaccid abdomen, repletion. He asked Dr. Ralfe for further information as to a case he described of albuminoid disease of the kidney, in which cardio-vascular changes supervened, remarking how rare these and albuminuric retinitis were in this affection of the kidney. He was rather surprised Dr. Ralfe had not attached greater importance to anæmia in the prognosis of chronic albuminuria. Personally he regarded it as a most important element in prognosis, and he thought one of the chief points in treatment was to obviate the anæmiating influence of chronic renal disease.

Dr. DE HAVILLAND HALL desired to comment upon a few of the points raised in Dr. Ralfe's most instructive paper. He thought that at the present time the tendency amongst assurance medical officers was to be too easy in passing lives with a history of albuminuria. He was of opinion that in young adults albuminuria might be a temporary condition, but he quite agreed with Dr. Ralfe in regarding albuminuria in persons over 40 as an insuperable bar to assurance. Dr. Hall alluded to several cases coming under his immediate observation of applicants for assurance with a past history of albuminuria, whose acceptance had entailed heavy losses on various companies. Like Dr. Ralfe and Dr. Stephen Mackenzie, he had watched with much interest the long period during which some patients with granular kidneys managed to survive and even to enjoy fair health. In particular he mentioned the case of an omnibus driver who was under his care as an out-patient for between ten and eleven years, and who was 76 years of age when last seen. He considered that one of the most useful parts of Dr. Ralfe's paper was that devoted to dietetic treatment, and that the principles therein laid down were in accordance with physiology and common sense. In endorsing the use of a strict milk diet in acute nephritis and in those forms of chronic nephritis attended with scanty urine, Dr. Hall referred to cases he had had under the milk treatment which had recovered, even after the supervention of grave uræmic symptoms.

Dr. HAIG was specially interested in the case mentioned by Dr. Ralfe in which coma and other severe symptoms of uræmia had somewhat unexpectedly cleared up, being replaced by an attack of acute gout. He had no difficulty in understanding such a case as he had elsewhere suggested, that uræmia was really due to an excess of uric acid in the blood. When it was in the blood it produced the well-known symptoms in the way he had explained; when it was driven out of the blood into a joint it produced acute gout. It was quite an easy matter to produce similar fluctuations in the solubility of urates, with similar but less severe symptoms. He pointed out that Professor Semmola, of Naples, had shown that in early and acute stages of Bright's disease there was an excess of diffusible albuminoids in the blood (hetero-albuminæmia), while in congestive albuminuria due to morbus cordis and other similar circulatory obstruction there was no such blood change. In this country, however, it was so difficult to get blood that the test could not be used very often. He was interested also in what Dr. Ralfe had said as to the incidence of albuminuria in patients taking morphine, for as he believed that Bright's disease was due to a general failure of metabolism, which

again was due to imperfect peripheral circulation, morphine, which in the period of abstinence exerted such a powerful effect in contracting the arterioles, might easily account for a little albuminuria, or in chronic morphinism even for Bright's disease.

Dr. SYMES THOMPSON observed that as our knowledge of the various forms of albuminuria becomes more precise (an inevitable consequence of such work as that of Dr. Ralfe), we are able to estimate with increasing accuracy the probabilities of life and to insure cases of established renal disease. There is really more difficulty in estimating the prospects in cases of undefined ill-health than in definite disease of kidney, lung, or heart. In the latter case a prognosis may be made with more confidence and precision than in the former, and extra ratings may be adjusted with greater confidence.

Dr. ROUTH would refer to Dr. Ralfe's able paper only in a gynæcological direction. The title of the paper was "On Chronic Albuminuria," and this did not necessarily restrict the albuminuria to renal disease. With women the mere examination of urine was sufficient, because uterine and vaginal disease frequently made it albuminous, as vaginitis, gonorrhœa, endometritis, ulcerations, &c. Then you might have albuminuria with hæmaturia (not to speak of the menses) from sanguineous endometritis, polypi, and fibroids, and even carunculæ of the urethra, and so on. In women, therefore, it was absolutely necessary often to make an examination, and in obtaining the urine to draw it directly from the bladder with a catheter. He knew of several instances, and these in high quarters, where, from motives of delicacy, examinations were not made, and a great blunder consummated. Then he was sure that in many of these cases where albuminuria existed and only a few blood globules were found, and where much gravel was passed, that the blood was due to the scraping of the mucous membrane by the rough crystals, and not to kidney disease. Lastly, in pregnancy, in ovarian dropsy, or where large abdominal tumours existed, albumen was often found, and even convulsions might occur during a labour, where all seemed due to pressure on the kidney and not disease in it. He would ask Dr. Ralfe if he knew of any *certain* mode of diagnosing between these several conditions. He, Dr. Routh, thought the presence only of casts could settle the question.

Dr. LAUDER BRUNTON thought that there could be only one opinion regarding the interest of Dr. Ralfe's communication. There were many points that might be discussed with advantage, but at that late hour he would only take up the question of albuminuria in relation to life insurance. In one of the New York insurance offices one out of eleven persons, apparently healthy, who applied for insurance was found to have albumen in the urine. The proportion here appears to be much smaller, and Dr. Brunton did not think he had more than about 2 per cent. At one time it was supposed by some of the New York offices that albumen in the urine of young people might not require any extra, but a number of cases having been followed up, it was found that their health deteriorated almost invariably. He thought, however, that there was a great difference in the prognostic significance of various kinds of albuminuria, and agreed with Dr. Ralfe, that albumen in the urine of men over 50 was a suspicious indication, although, if the specific gravity remained high, persons might live for many years. If the specific gravity was persistently low, the life was a precarious one, and frequently in cases of gouty kidney the albumen might be so slight as to be barely per-



ceptible. The test he usually employed for it was to acidulate with acetic acid and boil the upper part of it in the test-tube, allowing the lower part to remain clear as a standard for comparison. Frequently an exceedingly slight haze is all that is perceptible, even when the urine is looked at against a dark background. Persons in this condition might die very quickly after some gastro-intestinal disturbance brought on by over-indulgence in food, or exposure to chill, and he regarded the wearing of warm clothing a very necessary precaution. The albuminuria due to malaria was of much less prognostic significance than others, and he knew a man who was much above the average in mental and bodily power who has had it over twenty years. The peculiar complexion of the patient, looking as if the face had been brushed over with a thin coat of Indian ink or sepia, is often an aid in diagnosing such cases. In them the albuminuria was probably due to venous congestion occurring during the chills or during the vascular spasm which may occur either with or without a definite chill.

Dr. FRANCIS HAWKINS referred to the variability of albuminuria, and said that in cases giving every evidence of renal disease the albumen varied considerably in quantity. This was seen to occur irrespective of any special diet. There were also cases giving evidence of renal cirrhosis, such as frequent micturition, low specific gravity, and cardio-vascular changes, when no albumen was to be observed in the urine. A case was cited in which the urine had been examined at intervals for four years, and it was not until one year before death (which resulted from cerebral hæmorrhage) that albumen was found by the ordinary test. He thought the following cases might appropriately be mentioned:—CASE I. A. B—, aged 60, a business man, complained of “swelling of the feet,” in 1859. The only point of importance in the history was that when a young man the patient contracted syphilis. On examination the skin was pale, and there was œdema about the ankle, extending slightly up the leg. The apex of the heart was normal in position, and beyond a slight systolic murmur at the apex, there were no cardio-vascular changes. The specific gravity of the urine was 1010, acid, and contained nearly one-half albumen. Rest, with a restricted diet, was enjoined, and iron with cod-liver oil and a sojourn at Weymouth recommended. After being two months at Weymouth, the improvement in the general condition was marked, and his ordinary occupation, which necessitated a considerable amount of walking, has been followed up to the present time, while albumen is still present in the urine, and œdema of the ankles an occasional occurrence. CASE II.—W. D—, aged 56, a retired army captain, was seen in 1888. He made no complaint of illness, but stated “that something had been discovered to be wrong with his water when examined for life insurance.” Beyond malarial fever, he had suffered from no previous illness. He was a strong, well-built man, with very slight cardiac hypertrophy. There had never been œdema in any part. The urine was acid, specific gravity 1016, and contained a slight trace of albumen and uric acid crystals. Repeated examination always gave the same results. The case was regarded as one of early cirrhosis. It did not appear to be one of the cases of malarial albuminuria described by Dr. Lauder Brunton. A younger brother, aged 39, who had not suffered from malaria, also had albuminuria. In another case albumen had existed in the urine for twenty-five years. This case will be found fully reported in the ‘Clinical Transactions.’

Dr. NORMAN KERR, with reference to the alleged frequent presence of



albuminuria as a result of morphinomania, said his experience differed from that of other practitioners. Though he had, from the influence of traditional professional opinion, always been expecting to find albumen in the urine of morphinomaniacs, he had detected it only in a very small proportion of a considerable number of cases, probably in not more than 4 per cent. Where present there had generally been some pathological condition other than the morphine inebriety to account for the albuminuria. That very day, in a typical case of morphine indulgence of ten years standing in the person of a lady, aged 51, where he confidently expected to have found albumen, there was not a trace of it in the water. He, therefore, was inclined to the belief that, of whatever other pathological sins opium might be guilty, it was not, *per se*, a cause of albuminuria.

Dr. RALFE, in reply, agreed that when anæmia was pronounced the case assumed a very grave aspect, and no form of iron seemed to combat it successfully. As to the hæmaturia in gouty cases, he thought that the gouty poison might act like malaria, and cause abdominal plethora. The cases to which he referred of albuminuria associated with morphinism were all diabetics. In conclusion, he referred to two cases in which early transient albuminuria had been followed by serious symptoms.

---

*March 6th, 1893.*

## SUPRAPUBIC PROSTATECTOMY.

By G. BUCKSTON BROWNE, M.R.C.S.

PROSTATECTOMY means the actual removal, by knife, scissors, or forceps, of part of the prostate. At the outset of an inquiry into the merits of this operation it is desirable to have a clear idea of the anatomy and function of the prostate. For surgical purposes it is well to recollect that the prostate is only arbitrarily divided into lobes; it is really one single organ. As Ellis says: "Three lobes are described in the prostate, namely, a middle and two lateral, though there is no fissure in the firm mass." With regard to its function, I believe it is a sexual organ, and nothing more. To my mind it is quite sufficient to remember that in the female the act of micturition is perfectly well performed without a prostate, in order to reject all theories that it performs any urinary function. In adult and advanced life, in a certain number of cases, that is to say, exceptionally, the organ enlarges or hypertrophies, and by so doing frequently, but not always, interferes

with the act of micturition. This affection is met with in all sorts and conditions of men, and this has led to great difference of opinion regarding its causation, but it certainly does seem very frequently to follow sexual activity in middle and advanced life. I am more and more struck by the fact that prostatic hypertrophy often follows a second marriage, or a marriage contracted late in life. The results of youthful sexual excess are doubtless usually outlived. It is remarkable how rarely urethral stricture accompanies prostatic enlargement, and it may be presumed that men with urethral stricture have usually been sexually active in youth. When the prostate enlarges the hypertrophy may be (1) extravescical, or (2) intravesical, or (3) both extra- and intra-vesicular. It is the intravesical growth which chiefly causes difficulty in micturition. This intravesical growth is often like an egg projecting into the bladder, with the vesical urethral orifice at the apex of the egg. In such cases the projection is usually equal to an eighth, or a quarter, or even half of an ordinary hen's egg. This ovoid projection may be deficient at any part of the urethral circumference. When wanting anteriorly and laterally, we have the so-called middle lobe enlargement with which all are so familiar, where from behind the urethral orifice there is a projecting prostatic mass acting like a bullet valve, and often causing the bladder to be entirely dependent upon the use of a catheter for the voidance of its urine. More rarely we have the ovoid projection, only wanting in front, and we have a prostatic growth continuously surrounding the vesical urethral orifice on both sides and behind, or the projection may be only on one side; in such cases it is nearly always continuously combined with a posterior enlargement. While so rarely as practically never to be met with, the intravesical growth is only found anterior to the urethral orifice, intravesical prostatic outgrowths may be associated with considerable extravescical enlargement, and the latter may exist without the former, and cause the patient so afflicted to be partially or completely dependent upon his catheter.

In my opinion it is the intravesical growth alone which can be removed with reasonable safety, and with a fair prospect of recovery of the power of natural micturition. As, therefore, so much depends upon this form of hypertrophy, the answer to the question, Can we during life diagnose the existence of intravesical prostatic growth? is a very important one. Without digital ex-



amination after suprapubic cystotomy the presence of such growth can only be approximately diagnosed. In making this approximate diagnosis we must remember that we are dealing with a form of urinary disease where all unnecessary or excessive urethral interference is to be deprecated. It is precisely these cases which Sir Andrew Clark had in view when he so ably drew the attention of the profession to urinary fever following catheterism, or, as he called it, "catheter fever." This fever is unfortunately sometimes a fatal fever, and elsewhere I have attempted to prove that its severity is usually in direct proportion to the amount of urethral disturbance. The electric cystoscope, the use of which is never free from risk, is therefore particularly dangerous in the cases now referred to. In many its employment is impossible, or almost impossible, for there are cases in which the shape of the instrument is an absolute bar to its introduction, and in other cases the introduction is attended with so much difficulty that the very limited range of vision is obscured by blood. Several remarkably severe cases of cystitis which I have met with in practice, after endoscopic examination, where the prostate has been enlarged, have created a strong feeling in my mind against the use of this instrument in these cases. Again, the use of instruments which may be classed as urethrometers has not commended itself to me. They are all very trying to the urethra, which in this complaint we particularly desire to preserve from injury, and even when used they really do not appear to throw light upon this important point, whether there is or is not intravesical prostatic hypertrophy. There are, however, two simple means of examination, not trying to the patient and within the range of every surgeon, which, combined, will throw considerable light upon the question, namely, digital rectal examination and the careful measuring of the length of the urethra by means of the simple passage of the catheter. Any undue length of urethra will be an argument in favour of intravesical hypertrophy, and particularly so if by the rectum the prostate is felt but little enlarged. If much extravescical growth is found, this must be allowed for in estimating the importance of the length of the urethra. If the urethra is 9 inches long or more, and if not much growth can be felt by the rectum, there is almost sure to be intravesical hypertrophy. Should it be deemed desirable to sound the patient for stone, further evidence for or against the presence of intravesical growth will be obtained, for, if such



growth exists, it will be found that the sound has to travel very much up hill into the bladder.

Practically the consequence of all the forms of prostatic hypertrophy is almost invariably more or less difficulty and imperfection in the act of micturition. In some cases the prostate is enlarged without really causing urinary trouble, but these cases are rare. It is, however, most important to bear in mind that, even where there is great urinary difficulty, due to prostatic enlargement, the difficulty may be only temporary, due to congestion, and may pass away entirely in the course of days, weeks, and even months. This fact has an important bearing upon the question of prostatectomy. The following case is instructive in this connection:—

— aged 68: February, 1889. On a railway journey, owing to the presence of ladies, he did not leave the carriage. When arrived at his destination he could pass no water. Catheters were used unsuccessfully, and he was treated for suppression and not retention of urine. I saw him sixty-three hours after the last act of micturition, performed before starting on his journey. A catheter was passed and 3 pints of urine drawn off. The urethra was 10 inches long, and by the rectum the prostate felt enlarged. He recovered, but for three months all his urine was passed by catheter. Towards the end of April he began to pass water naturally, and in another month the retained urine—that is, water left behind in the bladder after a natural act of micturition—had come down to 2 ounces. In July the retained urine was only 1 ounce, and in February, 1890—a year after the retention—there was no retained urine. He is alive and well now, holds his urine from three to five hours, and does not use a catheter.

The explanation of the case is that the prostate was hypertrophied, but not sufficiently so to cause any real difficulty in micturition. The railway journey produced congestion of the already enlarged gland, and the confinement to the carriage without urinary relief caused over-distension of the bladder, producing temporary atony of its muscular coat. It would have been a surgical blunder to have subjected this patient to a suprapubic prostatectomy, from which operation he might easily have died. On the other hand, had he recovered from the operation, and recovered with full power of natural micturition, he would have been no better off, his life would have been risked, and prostatectomy would have obtained credit for what would have been brought about by far simpler methods. Such cases are by no means uncommon, and they are not unknown in the records of prostatectomy.

Unhappily, however, prostatic retention, whether partial or complete, generally comes prepared to stay, and we may next ask, What are the prospects of a man obliged to pass his urine entirely, or in part, by catheter? I would reply, that the prospect is a perfectly tolerable one, provided he becomes a master of the art. *Quoad* this particular complaint, there is no reason why he should not reach a good old age, and the chances are in favour of his not dying of his urinary complaint, but of some other disease or accident in the end. There is certainly nothing in his prospects to make him as it were lead a forlorn hope and rush into a grave and even dangerous operation such as prostatectomy at the beginning of his catheter life. Instances are numerous all round us of elderly men, active in all the pursuits of ordinary life, and many of them exceptionally highly placed in the conduct of affairs, who are obliged to pass all their urine by catheter, and who lead comfortable, useful, and happy lives. This state of comfort depends in the first place upon there being only a moderate amount of intravesical prostatic growth, and happily this is the rule and not the exception, and secondly upon the amount of attention paid by the patient to his condition. I shall not go into the details of this attention, but will only point out that such care consists:—(1) In the choice and proper skilful use of the best kind of catheter for the individual case; (2) the sufficiently frequent use of such an instrument; and (3) the immediate renewal of all instruments when they become cracked, rough, or otherwise worn. Also in (4) the use of an antiseptic lubricant; and (5) in the proper washing out of the bladder and application of medicaments to its mucous membrane in certain cases. All this may be spoken of as the proper toilet of the bladder. Taking everything into consideration, it is wonderful how tolerant the bladder usually is of neglect in many of the details of its toilet, but sometimes it is not tolerant, and neglect leads to such a state of vesical irritability, that is to say, a state of chronic cystitis, that life soon becomes a burden, because of the constant and imperious calls for the use of the catheter. This painful condition may also occur from causes over which the patient has no control, and which he cannot avoid by the greatest cleanliness and niceness in his self-management. One of these causes I would particularly refer to, for it is a condition which has almost escaped notice, but which has an important bearing



upon the subject of this paper, namely, the growth into the bladder of the prostate to such an extent as to act as a foreign body causing constant desire to pass water, and adding terribly to the trials of an elderly man already obliged to pass all his urine by catheter. Such a case I record in the 'Transactions of the Clinical Society,' vol. xxii, where I removed 4 ounces of purely intravesical prostate, giving the patient great relief. He is alive and well now, in his 92nd year, four years after the operation.

There are other cases where comfortable, or even tolerable, catheter life is impossible. In some very rare cases catheterism—I mean regular habitual autocatheterism—is practically impossible, owing to the extreme curve of the prostatic urethra forwards, the patient being supposed to be standing up, and, but not so unusually, there are cases where autocatheterism is so difficult that life under such conditions is not worth having. This difficulty may arise from the great curve of the prostatic urethra, the existence of false passages, and sometimes from the great ease with which the prostate will bleed if impinged upon and injured by the catheter, filling the catheter with blood clot, thus preventing the emptying of the bladder, and causing vesical spasm, as well as exhausting the patient by the constant loss of blood. Occasionally, also, we meet with patients whose hands are crippled by rheumatism, or by nervous affections, or who only possess one hand, or whose special occupations make autocatheterism difficult, or even almost impossible.

In all these cases where regular, habitual, autocatheterism is so frequent as to threaten to exhaust the patient, and where all palliative and other treatment has failed to bring relief, and in cases where, from the patient's urethral condition or want of manipulative power, such catheterism is impracticable, the question has long been—What are we to do? Up to 1886 we could only offer such sufferers a tied-in catheter, or a perineal, or a suprapubic vesical puncture or incision, followed by the permanent wearing of a tube through which the urine could drain away. Sir Henry Thompson, in 1874, devised a special instrument for the suprapubic puncture, and I published\* a description of an apparatus for these patients to wear after the suprapubic opening. This apparatus acts as an external receptacle for the

\* 'Brit. Med. Jour.,' August 4th, 1888.



urine, and patients can wear it and move about, fully dressed, with a fair amount of comfort. Many surgeons had before 1886, and have since, proposed the incision, and the excision of the intravesical prostatic obstruction, and also the burning of a groove in it by instruments introduced through the urethra, or by means of perineal incision, with the view of radically doing away with the obstruction, but no evidence has been brought forward which commends these operations to the surgical mind at large. The revival of suprapubic cystotomy, however, in the last decade suggested an attack upon the prostate from within the bladder from above, and independently of each other. Dr. Belfield, of Chicago, and the late and much regretted Mr. McGill, of Leeds, in 1886, became the pioneers in this important new surgical departure.

There is now no doubt amongst all surgeons that the obstructing prostate can be excised from above the pubes, but the surgical mind is not entirely satisfied that the operation, which is undoubtedly a severe one in itself, and necessarily performed upon those ill able to bear surgical interference, is justified because some doubt that even if the obstruction is entirely removed the bladder can regain its powers. Many surgeons believe that the prostatic hypertrophy is secondary to vesical changes, while others believe—and I do—that the prostatic enlargement is entirely primary. Sir Henry Thompson has expressed his doubts as regards the results of prostatectomy in the following sentences, and I need not say how weighty his authority is, and how clear and logical his mind. He writes in his *Clinical Lectures*: “I am entitled to require that if it does happen, or has happened, to any surgeon to divide or remove any part of an enlarged prostate for a patient who had previously been compelled to pass all his urine by catheter—say, for a period of twelve months—and that after the division in question he was enabled to dispense with the instrument, or at any rate to pass, say, only half his urine by natural effort, the case ought to be seen and examined by others. I desire extremely to see such a result from any of the proceedings alluded to. I have long wished to see this sight, and have travelled considerable distances abroad and elsewhere expressly seeking it, but at present without success.”

In answer to Sir Henry Thompson, I will briefly relate the following case; the patient is here to-night, and the facts are absolutely beyond dispute:—

R. H——, a gentleman, aged 70, tall and spare. He began to use a catheter in 1872, or twenty years ago. My first note of him is in April, 1886. All urine had then been drawn by catheter for four years. He now used his catheter seven times in twenty-four hours. The urine was clear and healthy. Catheterism was not easy; the instrument had to be well bent in order to go in. In 1887 he called on me complaining of bearing down of the rectum and much pain when he wanted his catheter. In May, 1890, he had bloody urine, and catheterism was necessary every hour and a half to two hours. On sounding his bladder the sound had to pass very much up hill in order to reach the bladder. During June the bleeding was so profuse that I feared a vesical growth. He was next seen in June, 1891. There was now great suffering and much vesical spasm and constant bleedings. He continued in this state until January, 1892, when I saw him with Dr. Cunningham, of West Hampstead. The catheter was necessary every half hour. There was considerable trouble from hæmorrhage and blocking of the catheter by clot. The urethra was  $9\frac{1}{2}$  inches long. By the rectum the prostate felt only moderately enlarged. On sounding him a small vesical calculus was found. All his pain was before catheterism; he had none afterwards until the next call for the catheter. His life was quite unbearable and perfectly miserable.

On March 10th, 1892, Dr. Frederic Hewitt administered ether. I could easily with tube and aspirator make the stone rattle against the former, but could not possibly seize it with the lithotrite, owing to the deep post-prostatic pouch in which it lay. The bladder was then opened above the pubes, and an intravesical prostatic subgrowth found, as large as a Tangerine orange, below and on both sides of the vesical urethral orifice. I removed the stone with a scoop—it resembled an orange pip, and weighed 9 grains—and then proceeded to remove all the prostate which projected into the bladder with forceps. Everything was twisted off, nothing torn or cut away. The pieces removed weighed 1 ounce. The bleeding was very severe; it was partially arrested by hot water injections, applied through the urethra by means of an enema syringe attached to the end of the penis. For forty-eight hours much blood came away with the urine. In three weeks some urine began to come by the penis. By the end of April the wound was closed, and all urine passed naturally by the penis, except 3 ounces which he drew off by catheter night and morning. Then came an attack of influenza in June, and the suprapubic wound slightly opened for a time, and we had some trouble in bringing a small suprapubic leakage to an end. Early in August all was perfectly healed, and by the middle of September we found the bladder able to empty itself to the very last drop, proved on several occasions by passing a catheter after a natural act of micturition, and no catheter has been used since. Of his own accord he wrote, under date January 3rd, 1893, "It is fifteen weeks since I saw you, when you advised me to do without the catheter. I have not used it since. I can now go in the daytime on the average three hours, and at night from four to six hours; indeed the last two nights I went fully nine hours each night; and this has happened twice before during the last three weeks. I cannot express the relief I feel from the dreadful pain and hæmorrhage I had so long endured: now I have not even inconvenience. I can now go about with more comfort than at any time during the last ten or twelve years; indeed it is twenty years since I first had to use the catheter."

This case speaks for itself. The patient had used a catheter



for twenty years, and for ten years had made no water except by catheter. He submitted to prostatectomy, and had full, natural power of micturition restored to him, an unspeakable boon, but to obtain which he undoubtedly risked his life. Such a case proves, beyond all question, that the habitual use of the catheter does not necessarily permanently destroy the power of the bladder to empty itself, as has been asserted upon great authority, and also proves that prostatic enlargement is the cause, and not the result, of troubles in micturition. With regard to the dangers of prostatectomy, I am just as sure that in a fatal case of suprapubic lithotomy in my practice the result would have been different had I left the projecting prostate alone, as I am that in several others the successful results were largely due to my non-interference with the prostate, although in several instances it offered itself temptingly for removal.

With reference to the details of the operation of suprapubic prostatectomy, I believe that the operation should always be extraperitoneal; all intraperitoneal operations must add largely to the risk, and all that is necessary can be done through an extraperitoneal incision. The bladder should always be opened upon a staff; if not—since the body of the prostate in these cases is frequently very large—it is very likely to be incised, instead of the bladder, when the surgeon thinks he is puncturing that organ with his knife, and mischief is done. The presence of a staff prevents such an accident. I believe in removing the projecting prostate with forceps and twisting it off in one piece if small, piecemeal if large. By twisting or torsion hæmorrhage is lessened. The intravesical growth should alone be attacked; no good will come of trying to remove the lateral lobes, even in part. In clearing the vesical urethral orifice of all surrounding projecting prostate tissue much assistance will be obtained by the presence of a metal sound in the urethra. Hæmorrhage is usually very free, and will be all the more so if the prostate has been attacked with knife or scissors, the use of both of which instruments I deprecate. Dr. Keys, of New York, has had much experience of prostatectomy, and has suggested a lint tampon drawn into the prostatic urethra by a thread through the urethra, but I have found the bleeding sufficiently controlled by hot water irrigation, and all plugs must add to the patient's pain. My space is limited, and I make no special reference to perineal prostatectomy as compared with



suprapubic, because the former operation is performed, practically and metaphorically, in the dark. The parts concerned are almost always beyond the reach of the finger, hæmorrhage is usually severe, and the risk of that most distressing condition, a perineal urinary fistula, resulting is very appreciable. I am also obliged to omit noticing the results obtained by the suprapubic route by Belfield, McGill, Mayo Robson, Jessop, Atkinson, Keyes, Southam, and Moullin; their works, however, speak for themselves.

In conclusion, I would express my present opinions as follow:—

1. Suprapubic prostatectomy should never be undertaken at the outset of catheter life unless regular catheterism is impossible.

2. The operation should never be undertaken as long as the ordinary catheter life is a tolerable one.

3. If, from any of the causes I have detailed, catheter life becomes intolerable, suprapubic cystotomy should be resorted to. By means of this proceeding the bladder can be thoroughly explored, and any stone removed, which in these cases may easily have escaped detection by the more usual methods of examination. The intravesical growth, if it is found to exist, and of this existence we can never be sure until the finger is in the bladder, can be fully examined, and removed if the operator thinks right to do so. If he deems removal inadvisable, or if there is nothing which can be removed, he can leave the patient with a suprapubic tube, for permanent after-wear, with the certainty that he will have materially improved the condition of the patient.

4. Should the operator decide to remove the prostatic obstruction, there is a very good prospect, but not a certainty, of the power of natural micturition being restored to the patient.

The PRESIDENT alluded to the impartial character of the paper, and regarded the conclusions as both wise and safe. He agreed that it was not advisable to interfere with anything more than the intra-vesical portion of the growth, though Mr. McGill had undertaken the removal of a great deal more. Some excisions of the middle lobe had been performed accidentally during lithotomy, when portions of prostatic growth lay in front of the stone, and had been pulled out by the forceps as the calculus was being extracted. He himself had once done this, and as he found that other portions of growth were removable he cleared the whole floor of the bladder. This was done five years ago, and the patient had made a perfect recovery. He agreed that enlargement of the prostate was a cause, and not a consequence, of bladder disease. He held that the old-fashioned silver catheter was a bad instrument for routine use, and that a

rubber catheter was much the best, the patient easily introducing it himself, and an upward curve of the urethra not interfering with its introduction.

Mr. REGINALD HARRISON did not desire to take this opportunity of pitting, so to speak, one operation against another, but he would like to ask Mr. Browne if he proposed to limit the operative treatment of enlarged prostate to supra-pubic prostatectomy. The circumstances calling for such operations were comparatively rare and exceptional. Where insuperable difficulties to the use of the catheter were caused by intra-vesical growths there could be no doubt that the supra-pubic operation was the best. On the other hand, there were instances where the hypertrophy took the form of a collar or bar encircling the neck of the bladder, which completely obstructed both the passage of urine and catheters. The supra-pubic operation was not applicable to the latter class, but great relief had been afforded by perineal prostatotomy. This operation, as elsewhere described,\* consisted in the division of the constriction from the perineum, the use of a properly selected drainage tube, and the maintenance of the increased space thus provided. Reference was made to eighteen cases where this operation had been practised by Mr. Harrison, not including instances where the supra-pubic method had, for reasons already stated, been selected. In ten of these cases normal micturition was either wholly or partly restored, whilst immediate relief to pressing symptoms was obtained in all. Dr. Belfield's† statistics, which included 133 instances, showed the perineal method as having a much less mortality than supra-pubic prostatectomy.

Mr. BRUCE CLARKE, referring to the question of diagnosis, said that the enlargement might lie either in the course of the urethra, at the neck of the bladder, or in the bladder itself. If within the organ, the growth should always be removed. With a bimanual examination under chloroform one might get a very fair idea of the size of the prostate. The most difficult cases of all to deal with were those in which the urethra was compressed by an upgrowth from its floor. After making a supra-pubic opening, the portion of prostate extending into the bladder might be, in some cases, successfully treated without removal by touching it with the galvano-cautery or with Pacquelin's instrument. He held that twisting out portions of prostate courted hæmorrhage, but that if the growth were got away by degrees, and the base touched with the cautery, much blood need rarely be lost.

Mr. SWINFORD EDWARDS remarked that the observations of the author with regard to prostatic hypertrophy being due to sexual excess after middle age were interesting, as tending to support the view that this organ is a part of the generative rather than the urinary system. He considered that the best means for ascertaining the existence of an enlarged intra-vesical lobe was by means of a short-beaked sound, supplemented with a finger in the rectum. Mr. Edwards also drew attention to the fact that hypertrophy of the prostate sufficient to cause total obstruction to micturition might arise even before 50 years of age. He had performed supra-pubic prostatectomy for the removal of a sessile outgrowth from the prostate in a man, aged 51, which totally prevented normal micturition.

Concerning the case of R. H—, upon the successful issue of which Mr. Browne was to be congratulated, it appeared to him that the supra-pubic

---

\* 'Transactions International Medical Congress,' 1884.

† 'Amer. Journ. Med. Sci.,' November, 1890.



incision was really undertaken for the removal of the stone, the prostatectomy being an after-thought, though a very proper one. It was a well known fact, in spite of what some authorities had said to the contrary, that a bladder which had lost all power of expelling its contents, even for several years necessitating habitual catheterism, might recover itself completely as soon as the obstruction was removed. This had been the primary result in four cases in which he (Mr. Edwards) had performed prostatectomy by means of Gouley's prostatome. For his part, he would be inclined to limit the operation under discussion to cases in which there was a distinct tumour growing into the bladder—sessile or pedunculated. In cases where the growth was more general, forming the so-called collar-like enlargement, he considered the risk of hæmorrhage by shock too great. Contrary to the opinion of the author, he was of opinion that where this operation was to be undertaken at all, it should be done as early as possible after the intra-vesical growth has been diagnosed (*i.e.*, before the kidneys had become affected, and also before the patient was worn out with pain and suffering), for the chance of a successful issue could not fail then to be much greater.

Mr. SHEILD asked whether silver catheters should be used at all in prostatic cases. He regarded them as dangerous instruments, especially if used on the first occasion when a catheter was required. The prostate varied much as to its vascular condition, and was apt to be much engorged when a stone was present. Very severe hæmorrhage followed in the only two cases of prostatectomy he had witnessed. Everything depended on a proper selection of cases. Bad subjects were old and fat men, especially those with bronchitis and emphysema and those with kidney trouble, whether that were indicated by the presence of albuminuria or by the passage of an excessive quantity of urine with a low percentage of urea.

Mr. BROWNE, in reply, said that in many cases in which growths or tumours had been removed by the perineum by accident in the blades of the stone forceps, though the wound had healed, the power of the bladder had not been restored. That might, however, be due to the operation not having been performed in a scientific manner. In the case alluded to by the President, there had been a long-continued urinary fistula. He admitted their indebtedness to the President for introducing the vulcanised rubber catheters, which were undoubtedly the best in prostatic cases, but it could not be denied that the rubber catheter did not always "go." There were two catheter traps at the very neck of the bladder. These were the prostatic sinuses, one on either side of the veru montanum. When the middle lobe was enlarged, these sinuses were deep posteriorly, and formed a very complete obstruction to soft instruments. Though Mr. Harrison had been fortunate in respect of hæmorrhage and urinary fistula, his own experience had not been by any means so satisfactory. Moreover, this unsatisfactory experience had not been his alone, but he had observed it in the hands of others. Statistics were valueless in respect of this operation, their number being comparatively small, and the individual cases differing so much from each other. Keyes, of New York, described it as "the most horrible operation in surgery." He agreed that, as a general rule, the use of silver catheters was not advisable, but at the same time there were cases in which they were indispensable in order to effect an entrance into the bladder. As to kidney disease, he observed that these were desperate cases, and the surgeon could not stop to weigh possibilities in endeavouring to afford relief.

---



*March 13th, 1893.*

## THE TREATMENT OF LUPUS OF THE FACE BY FREE REMOVAL AND SKIN GRAFTING WITH LARGE FLAPS.

By W. BRUCE CLARKE, M.B., F.R.C.S.

It is nearly 300 years since Tagliacotius published his classical work on the restoration of lost parts by the transplantation of large skin flaps, but it is only within the last twenty that surgeons have been able to shake themselves free from the belief that the skin flap must remain partially attached to its original site until the connection with its new surroundings was firmly established.

It was only in 1871 that Reverdin published his now well-known method of taking minute grafts of skin and attaching them to a granulating surface. Four years later, viz., in 1875, Wolfe, of Glasgow, described a plan of removing large flaps of skin, which, though it is successful enough when thoroughly carried out, is somewhat tedious of application, and has never received that recognition which it rightly deserved.

In 1888, at the German Congress of Surgeons, Thiersch, of Leipsic, brought forward a plan of skin grafting which combines the merits of simplicity and ease of application, and with moderate care usually yields a successful result. It is applicable either to fresh wounds or granulating surfaces, and it is not too much to say that it has revolutionised the treatment of certain affections and certain surgical conditions. There is hardly any limit to the area which such skin flaps may be made to cover, whilst inasmuch as the whole thickness of the skin is not removed from that part of the body from which it is taken, but slight chance exists of adding a new deformity in the attempt to remove an old one. I showed several years ago at one of the Societies a child the greater part of whose skin had been completely torn off between the knee and the ankle by a tramcar accident, but by the aid of skin grafts obtained by this method and placed upon the granulating surfaces complete healing had taken place with an excellent and useful leg.

For the purpose of operating, all that is necessary is a large broad razor and a small bowl of warm water. The part of the body selected, usually the front of the forearm or the inner side of the thigh, is then carefully washed and rendered as aseptic as possible, after which strips of skin are cut off with the razor and dropped into the bowl of warm water. With practice, strips may be easily cut 4 or 5 inches in length and an inch in width. The cut must not traverse the whole thickness of the skin, so as to ensure sufficient being left behind to restore the surface portions that have been removed. Experience has shown that it is preferable to use the patient's own skin so as to avoid all possibility of infection or the transmission of such diseases as syphilis, tubercle, &c. In the case of skin grafting for lupus, it is of course unnecessary to employ such large pieces of skin as those above described, but, if success is to be looked for of a permanent character, all the lupoid tissue should be first freely removed, not only with a spoon, but with a knife as well. Anyone who has ever made the attempt to scrape a patch of lupus cannot fail to have observed that the granulation tissue which covers the floor of the ulcer and the friable edges which surround it are removable with the greatest ease, but that as the sounder tissues are reached more and more difficulty is experienced, and eventually the sclerosed surroundings offer even more resistance to the Volkmann's spoon than does a piece of healthy skin. Having had under my care a boy with a lupus patch, which I had several times scraped with only temporary success, and being anxious to ascertain whether the removal of all the softened tissues was identical with the removal of all the disease, I determined on the next occasion to remove layer by layer with the scalpel, examining each of them by frozen sections at the same time. My friend and colleague, Mr. C. B. Lockwood, who was at that time surgical registrar, kindly brought the freezing microtome into the ward, and each layer as it was removed was frozen, cut, and submitted to microscopical examination by him. Stated briefly, our investigation proved without any doubt that the hard sclerosed tissue at the base of the ulcer contained numerous little pits in it which were full of cells, some of which were undergoing degeneration, and it was not until all this hardened tissue was removed and structures were encountered which looked healthy to naked eye inspection that they proved likewise to be healthy under the micro-



scope. Tubercle bacilli we did not find because our microscopic examination was necessarily somewhat rough-and-ready, but I certainly satisfied myself, and I have every reason to believe that Mr. Lockwood agrees with me, that all the hardened tissue must be removed and the skin for some  $\frac{1}{8}$  inch or more outside the margin of the ulcer if the tendency to return is to be completely obviated. It goes without saying that such a mode of procedure involves very free venous and capillary oozing, and the arrest of this forms one of the principal practical difficulties with which one has to deal. A pair or two of pressure forceps, plenty of very hot sponges, and a goodly supply of patience are, however, all the equipment needed, but I have had to keep up pressure with hot sponges occasionally for as much as half an hour before the hæmorrhage was sufficiently arrested to warrant one in placing the pieces of skin on the exposed surface. In my later cases experience has shown me, however, that the skin flaps themselves are important adjuncts to the arrest of hæmorrhage, and thus one has learnt a means of considerably shortening the operation. As soon as the skin has been cut from the leg or arm, as the case may be, it curls up almost like a shaving, and when placed upon the ulcer requires to be unrolled and adjusted with a couple of needles, and with almost as much care as one employs when mounting a thin section for the microscope. The edges of these large skin grafts should always come well up to, if they do not absolutely overlap, the edges of the ulcer which is being grafted. When careful adjustment is completed, a little iodoform or other dry powder is dusted over the part, and two or three layers of dry gauze superimposed. About a week later the dressing is removed, when complete union will usually be found to have taken place. If this is not the case, as soon as the unhealed part of the ulcer is in a granulating condition, a fresh graft can be applied in a precisely similar manner.

It occasionally happens that the ulcerating process has spread round the margin of the nostril and is invading its interior. If this is the case a small iodoform gauze plug must be gently inserted into the nostrils, in order to keep the skin graft in its place. Indeed, whenever the lupus is situated near the orifice of the nostril, it is well to keep the nostril occluded by gauze, in order to facilitate the healing process by keeping the parts dry. In one of the patients shown here to-night it will be seen that a portion of one of the lateral cartilages of the nose had disappeared before



skin grafting was attempted, but this did not in the least interfere with the healing process. Any one who has witnessed the great difficulty which is experienced in getting a patch of lupus about the nose which is as large as even a sixpence soundly healed by ordinary methods, will easily realise the advantage which is afforded by the method I have just described. I may say, in conclusion, that I have had seven cases of lupus of the nose under my care which have been treated by this method, and not one has failed to heal kindly from the first. Two cases are here exhibited to-night, one of which was treated by me just a year ago and the other a few weeks back. In none of them has there been any tendency to ulceration of the flap. In one or two there has been round the edge of the old ulcer a slight return of the lupoid tubercles, but these have readily disappeared when touched either with a galvano-cautery or with nitric acid. Had the edges of the ulcer been removed a little more freely they would probably never have returned at all.

*The following cases bearing on the same question were then exhibited by the following gentlemen :—*

Mr. BRUCE CLARKE showed two cases: the first was that of a young woman on whom he had operated about eleven months before, removing the lupoid tissue by excision, baring the cartilage of the nose and clearing out the diseased mucous membrane from the interior of the nasal cavity. The second case was also in a woman, and was one of lupus of the nose of a more exuberant character. In this, owing to failure to excise the growth at the margin, there had been a slight recurrence of the disease.

Mr. WATSON CHEYNE showed a case with the following history :—Two years previously the patient had been treated with tuberculin. When he first came under notice he had extensive lupus of both cheeks, neck, nose, and upper lip, also on back of forearm and hand. After two injections the lupus patches on one cheek were scraped, but the nose and lip were left untouched. After about five months' vigorous treatment with tuberculin, patient was sent home apparently perfectly well, the parts being beautifully smooth and soft. A year ago he returned with a number of spots of recurrence on the right side of the face and on the nose and neck. The whole of the affected area of skin was excised, the incision extending all round in the healthy for from  $\frac{1}{4}$  inch to  $\frac{1}{2}$  inch

beyond the affected part. The raw surface left was completely covered with skin grafts. At the same time the patch on the back of the hand was similarly treated, a number of extensor tendons being left bare. A fortnight ago he returned with recurrence on the left side of the cheek, which five days ago was excised and skin grafted. As to his present condition, the upper lip, which was treated with tuberculin, alone remains well; the skin-grafted cheek and nose of a year ago remains sound, and is assuming a natural colour and softness; the recently applied skin grafts have almost entirely taken.

Mr. BIDWELL showed two cases on whom he had operated in a similar manner to Mr. Watson Cheyne. The first was a girl, 18 years of age, who had suffered from a patch of lupus, measuring 3 by 4 inches, on the inner side of the left thigh. It was dissected off and the surface grafted by Thiersch's method. The second was a woman, aged 35 years, who for eighteen years had suffered from lupus over the right shoulder. Its superficial area was, at the time of operation, nearly 30 square inches. The whole thickness of the skin and subcutaneous fat were dissected off, and the surface covered with Thiersch's grafts. Both cases were soundly healed. Mr. Bidwell recommended that in long-standing cases the subcutaneous fat should be removed in addition to the skin, since it is found to be altered in consistence in these cases.

Dr. RADCLIFFE CROCKER said that the method at present in vogue was as great an advance upon scraping as scraping had been upon previous methods. For the last two years whenever he had dealt with a small patch he had practised excision. Mr. Barker's plan to prevent the graft curling up was a good one, viz., laying it on a sponge and then applying it to the raw surface, in the same way that a gilder used gold leaf.

Mr. BALMANNO SQUIRE said that he remembered Mr. Durham exhibiting, very many years ago, at one of the societies, a patient on whom he had recently operated by excising a patch of lupus from the left cheek. In the case referred to, cicatrisation was favoured by curving strips of skin, borrowed from the circumference, over the raw surface, namely, towards its centre—Catherine-wheel fashion. The treatment of lupus by excision extended back to a comparatively remote period. The modification of the old operation of excision, as now proposed, left still untouched the as yet extremely difficult, and always very important, question of how to deal successfully with lupus of the cavities of the mouth and nose. Koch's tuberculin, from what he had seen of its effects, had also failed absolutely to solve this question. He had examined carefully all of the cases brought forward that evening illustrating excision. The excision involved not only the removal of the entire thickness of the affected skin, but, as had been admitted, some of the subcutaneous fat also, and, in addition, what appeared to him a somewhat gratuitous quantity of the surrounding sound skin—as much as a  $\frac{1}{2}$ -inch radius of it in some of the



cases. It seemed to him that this operation necessitated needless mutilation, while on the other hand it did not appear to be so thorough in its effect as to claim justification on the ground of thoroughness. He noticed in some of the cases patches of very unquestionable lupus, which were by no means of very minute size, around the cicatrix left by the operation. Then as to the question of mutilation. One of the patients, whose cheek had been operated on, presented in consequence a very fallen-away and slab-like appearance on that side of his face. Two other of the patients who had each of them been operated on as to one of the wings of the nose presented, as he thought, a needlessly mutilated appearance, although they had evidently been operated on with very great skill. As to the demerits, if any, of the operation of scraping away lupus with the steel spoon, he believed that this operation had been discredited unduly, and for four principal reasons. Firstly, the spoons used were a great deal too large. Secondly, they were a great deal too blunt. Thirdly, they were handled in a manner unsuitable to the purpose in view. Fourthly, as soon as the operation was commenced the bleeding, and not only that but also the consequent blanching of the skin around, obscured completely the map of the diseased patch during the operation. Shortly after the first introduction of scraping into this country, now very many years ago, he had introduced an improved form of steel spoon which was very much smaller and very much sharper, and provided with a much slenderer handle than the original German models. By means of this improved spoon, owing to its smallness, the sinuosities of the circumference of a patch of lupus could be more accurately followed, and also minute, isolated patches could be more readily scraped out. Owing to the greater sharpness of the spoon, the scraping would, moreover, be much more thoroughly performed. Then as to the method of handling. The German spoon, which had a large and thick handle, was grasped in the clenched fist and used with a pushing movement, that is to say, in a direction from, and not to, the operator. This method of handling the spoon was very unfavourable to the accuracy demanded for a successful result. His improved spoons were held in the same way as a pen or pencil, and he had found this method of handling much more conducive to accuracy and to efficiency. Then again, in order to avoid the limits of the lupus patch becoming lost to view during the operation, he had introduced the practice of carefully marking out the patch before the operation by some indelible means so that the map of the patch could be kept in view during the whole of the operation. Many years ago he had devised and introduced the practice of linear scarification. In cases where careful and thorough scraping had been followed up by adequate linear scarification, he had seen much better results ensue than were presented by any of the cases exhibited that evening; that is to say, very considerably less mutilation was caused than in any of the cases now shown, and yet a more thorough result was obtained than was visible in the majority of the excision cases now before the meeting. When the ala or lip of the nose was affected (and these places are the commonest situations of lupus) he had found linear scarification alone, without scraping, much less mutilating than with the addition of scraping, and still less mutilating than excision; but yet, certainly, quite as efficient as excision, that is to say, absolutely efficient. It was true that linear scarification when employed alone needed to be frequently repeated before complete recovery was attained; but scarification had the effect of causing the cells of the lupus tissue to undergo metamorphosis into fibrous tissue, and so was more conservative than any



other known method of treatment. The preservation of the nose, when possible, in its normal shape was a matter of extreme moment to many patients, and was worth a good deal of trouble and patience. From what he had seen of the results of excision, as exhibited that evening, he greatly preferred the results which, with due care, were obtained by scraping and linear scarification. But still he thought that the meeting ought to feel very much indebted to Mr. Bruce Clarke for having brought the matter before the Society.

Mr. C. B. LOCKWOOD said that the results which had been obtained were better than those usually seen after treatment by ordinary methods. The tissues far beyond the seat of growth, whilst looking apparently healthy, would often be found on microscopical examination to show the cell infiltration characteristic of the disease.

Dr. SOLOMON SMITH remarked that at the present time the treatment of lupus seemed to lie between erasion with subsequent dot-grafting and the method of excision with immediate sheet-grafting which had just been described, and he thought no one could look at the admirable results which had been shown without admitting that the latter method was the best. It seemed to him that the recurrences of lupus generally occurred in places where the scar was stretched and irritated by the contraction of cicatricial tissue, and that, while no doubt one great advantage of the new method of sheet-grafting was the latitude it gave for wide removal of all diseased structures, another and no less benefit arose from the fact that the immediate covering of the raw wound by a sheet of living epidermis prevented the formation of granulation tissue and the subsequent contraction of the scar. This was a great advance on the system of dot-grafting on the surface of a layer of granulations which were certain afterwards to contract, and one of the best points in the cases shown was, not merely the completeness of the removal, but the elasticity of the scar left by the operation.

Mr. BRUCE CLARKE, in reply, said that the recurrence in one of the cases was due to the fact that he had not removed the skin surrounding the ulcer sufficiently freely. By injecting tuberculin beforehand a surgeon might be able to better define the limits of the outlying patches, which without it were often invisible.

## TWO CASES OF ABDOMINAL SECTION FOR TUMOURS WHICH PRESENTED UNUSUAL CHARACTERS.

By W. H. BATTLE, F.R.C.S.

THE cases of which I read notes to-night present many features which are in my opinion of exceptional interest. They had not much in common; they were, however, both abdominal tumours, probably of ovarian origin, and were unusually movable, but differed altogether in their pathology, in their history, and in the symptoms to which they gave rise.

A married woman, aged 41, came to my out-patient department at St. Thomas's Hospital on the 19th of July of last year, com-

plaining of a tumour of the shoulder, also of a lump in the abdomen, tender on pressure, and was on the same day admitted to the Elizabeth Ward.

The family history was as follows :—Her father died at the age of 53 of consumption. Her mother, aged 75, was in good health, but had undergone some operation on the bladder (the nature of which the patient did not know) seven months before. There were fourteen in the family, ten of whom were living and well. She had had six children : five were alive and strong, one was stillborn.

She had always been strong, and had never had any serious illness. About fourteen years before she had an attack of “colic” which lasted about a fortnight, and she used to be subject to bilious attacks. The bowels were usually constipated ; the catamenia regular. The tumour on the shoulder had been noticed about eight years, during which time it had only increased slightly in size ; there had been no pain in it except on lifting heavy weights. She was unable to lie on that side, as the position caused the tumour to ache.

The tumour in the abdomen had been noticed about nine months. There had been no pain in connection with it. Its position was in the right lumbar region at first. At times it would almost disappear. When its position changed the patient said she felt very sick, but did not actually vomit ; she would also feel hot and trembling. It was tender on pressure, and she did not think there had been any increase in its size.

The general condition of the patient was good ; there was no albuminuria or evidence of visceral disease. The tumour of the shoulder was a lipoma as large as the palm of the hand.

The tumour in the abdomen was situated in the right iliac fossa, being more easily felt at some times than at others, though the patient could always readily find it herself. It was rounded, smooth on the surface, about the size of an orange, flattened, and tenderness was complained of when it was manipulated. It could not be fixed sufficiently to permit one to say that there was any fluctuation in it. The most marked character which it possessed was its extreme mobility. Usually found, as I have said, in the right iliac fossa, it could be carried into either lumbar region, upwards under the liver and even into the left hypochondrium without any very evident resistance, but always returned to its original position.



On the 22nd of July Dr. Cullingworth kindly examined the woman, and reported that this tumour appeared to be unconnected with the uterus or appendages, which were apparently normal.

On the 1st of August the patient was placed under the influence of ether and a median longitudinal incision of about 2 inches in length made between the umbilicus and pubes. A few bleeding points having been secured, the peritoneum was opened and the edges held aside with forceps. The tumour, when brought under the incision, was found to be fluctuating, and of a yellowish appearance. It was punctured with a trocar and cannula, but no fluid came through the instrument, but around the point of puncture a thick, whitish material came to the surface and was wiped away. Absorbent sponges were then packed around the opening, which was enlarged, and the contents evacuated by pressure on the cyst. When the size of the tumour permitted of its being brought through the opening it was placed on a large sponge and separated from its attachments. These were three in number, to the omentum by means of a thick broad band, to the right ovary, which was small, by a thin band, and to the large intestine by a long pedicle. The attachment to the omentum was such that the tumour was partly surrounded by the free border of that structure. The wound and parts exposed were cleansed with warm boracic solution and the wound closed with interrupted silk sutures. The left ovary was healthy. The contents of the cyst consisted of sebaceous matter and hair. There was a rise of temperature the same evening to  $100\cdot4^{\circ}$ , and on the following evening to  $100\cdot6^{\circ}$ . On the third evening it was  $99\cdot8^{\circ}$ , and afterwards normal. The subsequent progress of the case was uneventful, and she left wearing an abdominal belt on the 20th August.

The opinions given as to the nature of this tumour were many and differed considerably—movable kidney, floating spleen, sub-peritoneal pedunculated fibroid, ovarian with a long pedicle, lipoma of omentum, and dermoid cyst of omentum.

Perhaps you may remember a case under your care, Mr. President, in which a tumour of very similar character was removed by Mr. Sydney Jones from a patient who had been in your ward. The patient, a woman, aged 35, was under treatment in 1884, and the cyst in that case grew from the wall of the small intestine. The most marked symptoms in connection with it were excessive



tenderness and extreme mobility. I showed the specimen at the Pathological Society in the following January ('Trans. Path. Soc.,' 1885, p. 213). Its usual situation was in the left hypochondrium. My recollection of this case induced me to think the tumour possibly a dermoid of the omentum, a small lipoma of the omentum, or a small ovarian with a long pedicle, but I was in favour of one of the former, as I did not recollect any case of ovarian tumour which had been described with a pedicle of such unusual length. It seemed to me that the comparative absence of symptoms and the ease with which it could be moved from one place to another, though far distant, as well as its shape, negatived any diagnosis of movable kidney or spleen.

The second case which I wish to bring before your notice to-night is that of an unmarried woman, aged 21, who was admitted under the care of Dr. Payne in the "Christian" Ward of St. Thomas's Hospital on the 28th of June of last year, complaining of a swelling of the abdomen.

The family history was good. She had been laid up eleven years ago for what she said was an abscess of lung which broke through her side, and was obliged to keep her bed eleven months. About three months before she came under treatment she first noticed that the abdomen was swelling, and she had increased shortness of breath, but had not suffered from palpitation. She had brought up blood by vomiting a month ago, perhaps half a small teacup full. Otherwise she had felt well, though she was suffering from a slight cough.

She was on admission a somewhat anæmic girl complaining of "dropsy of the stomach." On examination there was no evident dyspnoea, the chest moved badly, the costal angle was wide, and the lower ribs protruded. In front the resonance ceased at the 4th rib on the right side, and in the axilla about 4 inches from its apex. Breath sounds normal. On the left side resonance was also good but ceased at the 3rd interspace in front, and laterally 4 inches from the apex of the axilla. The inspiration was distinctly harsher at the left apex than on the right side. Posteriorly the resonance ceased on both sides at the level of the 8th rib. The apex of the heart beat just above and very slightly internal to the left nipple. The dulness above began at the 2nd rib. At the apex a loud diastolic murmur was heard conducted into the axilla. With this a faint systolic murmur was also audible. A double

murmur, of which the systolic was the louder, was heard over the aortic area. A double murmur was also heard over the left edge of the sternum. A marked impulse could be seen over the intercostal spaces at each beat of the heart. The pulse was 92, regular, and inclined to the water-hammer type.

The abdomen was much distended; no enlarged veins were visible; a fluid thrill could be easily obtained. There was resonance over an area of about 4 inches in extent below the ensiform cartilage and also in the right flank. Liver and spleen not felt and no tumour detected. A thrill could be felt from one flank to the other. Girth,  $36\frac{1}{2}$  inches. There was a large soft fluctuating ovoid hernial swelling in each labium majus, the contents of which could be returned into the abdomen by pressure; only slight impulse was felt on coughing. I may here remark that these swellings were dull on percussion, the right was larger than the left, and that a third swelling was afterwards found of considerable size, which projected from the vulva and evidently consisted of a protrusion of the posterior vaginal wall. The tongue was slightly coated and the bowels somewhat constipated.

The urine was loaded with lithates, had a slight deposit of mucus, and contained a good trace of albumen. There was no œdema of the legs. The temperature was normal. She had had amenorrhœa for three months. On July 3rd it was noted that in the lower part of the abdomen a little to the right of the middle line a large hard mass could be felt. This was freely movable in every direction and could be displaced into the left hypochondrium. It also seemed to float in the fluid, and felt quite hard.

Dr. Cullingworth, who examined the patient on the 5th of July, reported that the cervix uteri was small and normal, the uterine canal of normal length, its direction upwards, backwards, and to the right side. No obvious connection existed between the hard tumour and the uterus.

On July 7th the albumen had disappeared from the urine.

When I saw this patient, at the request of Dr. Payne, the condition was much as described above, the abdomen considerably distended, bulging laterally, and she turned in bed with difficulty. A tumour apparently the size of a child's head could be felt, rounded and firm, easily displaced, a tap over it when it touched the left side of the abdomen being sufficient to make it travel to the opposite side, where it could be felt to impinge with some force,



and *vice versâ*. The hernia protrusions were large and presented a curious appearance.

On July the 16th, when the patient was transferred to the surgical side of the hospital, the girth of the abdomen had increased to  $37\frac{1}{2}$  inches, and the patient complained very much of the weight.

The operation was performed on the 18th at 2 o'clock. Chloroform was first tried by the anæsthetist, but as the pulse became slow and irregular this was discarded and A.C.E. mixture substituted. From this time her condition improved as the operation proceeded. A longitudinal incision about 2 inches in length was made in the median line below the umbilicus. After the few vessels which were in the abdominal wall which bled had been secured, the peritoneum was incised and the fluid in the peritoneal cavity, which was not of an ascitic character, but like the thick glutinous fluid of an ovarian cyst, gradually evacuated. The quantity of this fluid measured 10 pints 9 ounces. The tumour, which was freely movable in the peritoneal cavity, was brought under the incision, and as it was a multilocular cystic one, an attempt was made to empty some of the cysts by means of a trochar and cannula, but the fluid was too thick to pass through the cannula, although it was one of large size. The dimensions of the tumour being too great to permit of its removal through the small incision, this was enlarged to the required extent upwards and downwards. The tumour had had its origin in the left ovary and the pedicle was long and well developed; it was secured by means of two silk ligatures, one of them being brought round the pedicle and tied immediately below. The abdomen was cleansed as far as possible by means of warm sponges, and washed out with a solution of boracic acid at a temperature of  $102^{\circ}$ , and the incision closed by means of interrupted silk sutures. The herniæ which were so prominent a feature before the operation disappeared during the escape of the fluid from the abdomen, and one could easily pass two fingers through the internal ring on either side. The pouch of Douglas was much larger than usual, the posterior wall of the vagina lax and easily protruded. A curious condition was noted after the operation, in consequence apparently of the prolonged and uniform pressure of the heavy fluid—the small intestines and omentum remained in the upper part of the abdomen and showed no tendency to descend into the pelvis; accordingly a



large space was left in the lower part of the abdomen unoccupied by anything but air, across which the abdominal wall passed somewhat like the membrane of a drum.

After the operation there was some shock, for which a hypodermic injection of brandy was given. The temperature at 4 p.m. was, however, only  $95^{\circ}$ , but became normal an hour later.

The patient made a satisfactory recovery from the operation and left the hospital on the 24th of August wearing an abdominal belt. A simple enema was given on the 4th day and solid food allowed. The stitches were removed on the 8th day, but she was not allowed to get up for more than three weeks after operation. The temperature at no time exceeded  $99.2^{\circ}$ .

When she left the hospital she was wearing a double inguinal truss, before which a double spica bandage had been applied. A point of interest was noted for nearly three weeks after the operation, and that was the presence in the hernial sacs of a considerable quantity of air which could be easily pressed back into the abdomen. On the 17th day it had nearly disappeared from the right side but not from the left.

The tumour was an ordinary multilocular one, none of the cysts being very prominent, and presented at one part a circular opening about 2 inches in diameter where the cyst had ruptured, the edges of which were clearly defined, and almost in contact with the lining of the cyst, on its further side. It was difficult to imagine that the large amount of fluid which was evacuated from the abdomen at the operation could have been secreted by the lining of this cyst, for it would not have covered an ordinary orange, yet the fluid was like that of the ordinary ovarian cyst, not so thick as that in some of the cysts in the tumour removed, but as thick as that usually evacuated through a Spencer Wells' trochar.

This case presented several features of interest, and I will briefly mention some of those which appear to me the most important and worthy of your attention. The history of the case, gradual and painless enlargement of the abdomen, the presence of the large amount of fluid, and later the discovery of a tumour of apparently solid character in the fluid, made me think that it was probably a case of sarcoma of the ovary with secondary peritoneal growths and effusion into the peritoneum, so it was an agreeable surprise to find that the growth was of simple character and the

fluid due to a rupture of one of the many cysts composing it. The hernial protrusions and the protrusion of the posterior vaginal wall are so far as my experience goes extremely rare in the young when suffering from ascites, and perhaps these swellings with the sensation given me by the tumour, as if it floated, should have aroused suspicion as to the density of the fluid. The curious manner in which the intestines remained in the upper part of the abdomen flattened and emptied without tendency to descend in spite of sponging, and in spite of flushing with warm boracic solution, is very unusual, and the necessity of closing the abdominal wall over a large cavity containing only air was new to me. The observation permitted by the patency of the internal rings, and the existence of the inguinal sacs as to the length of time during which the air remained unabsorbed, although not of great importance, was still of much interest.

The albuminuria was probably independent of any renal disease, as it disappeared when the patient had been kept in bed for a few days and did not reappear.

As a rule in cases of heart disease which require operation I prefer ether as being generally the most safe anæsthetic, but if the anæsthetist is a man of experience the decision is left to him. I have alluded in the notes to the manner in which this patient took the anæsthetic; the valvular disease was extensive, and it is probable that the greater quiet in the early stage met with during chloroform administration was of value as a help in the subsequent administration of the A.C.E., there having been no unnecessary excitement or struggling.

Dr. CULLINGWORTH (speaking at the call of the President) said that both the cases brought forward by Mr. Battle were of exceptional interest. He was afraid his own connection with them was not altogether creditable, for in both instances he had been asked to examine the patients and give his opinion as to the nature of the swelling, and in both his opinion had been wrong. He certainly had been unable to satisfy himself that in either case the swelling was connected with the uterus or its adnexa. In the first of the two, he believed a diagnosis was next to impossible. With regard to the second, he should like to ask Mr. Battle whether the opening through which the contents of the cyst had escaped into the abdominal cavity appeared to have been caused by ulceration or by rupture.

Mr. BATTLE in reply, remarked on the serious nature of the cardiac complication in the second case. There was nothing to indicate the rupture of the cyst; the history was that of a simple ascites. He had no doubt that the intestines ultimately returned to the lower part of the abdomen and occupied their normal position.

---

*March 20th, 1893.*

## SYMPTOMS OF MENTAL DISSOLUTION.

By GEO. H. SAVAGE, M.D., F.R.C.P. Lond.

MR. PRESIDENT AND GENTLEMEN,—When first with a light heart I accepted the invitation of your Secretaries to contribute a paper to this Society, I felt full of hope that I might from my special experience contribute something of interest to the Society; this hope dwindled as time passed and material increased, and I now offer, I fear, a meagre contribution.

I began by collecting together the notes of all patients, over 60 years of age, who were admitted into Bethlem Royal Hospital during my seventeen years' physicianship there. I am free to admit that this collection proved little but a heap of facts which, however, revived old impressions and formed certain broad principles which I shall lay before you without statistical details, as I believe a broad sketch outline may more readily give a clear idea of my impression than would a definite picture.

The question of evolution has been so long before us, and in related subjects dissolution has also taken such an important place in world science, that I feel no apology necessary for introducing the subject, though I may for the manner of its treatment. Whether it was Goethe or Oken who, struck by the separating bones of a sheep's skull, was first made to see that the bones of skull and the bones of the spine were really the same, only modified, matters not; dissolution taught the lesson, and in any branch of knowledge similar lessons have been learnt from observations of the facts of dissolution, and I am confident that much of the future progress of psychology depends on the observation of dissolution of mind.

Of dissolution Herbert Spencer writes: "When an aggregate has reached that equilibrium in which its changes end, it thereafter remains subject to all actions in its environment which may increase the quantity of motion it contains, and which are sure



either slowly or suddenly to give its parts such excess of motion as will cause disintegration. The course of change in the dissolution so caused being the reverse of evolution, the illustrations of it may properly be taken in the reverse order."

(179) "In organic dissolution there is a transformation of the motion of the aggregates into the motion of the units. Death ends all the integrated motions of evolution, and the process of decay shows an increase of the insensible movements, in the fact that the gases generated by decomposition contain more motion than the matters from which they arise."

(180) "The waves rolling about the small pieces of the undermined cliff, and in storms turning over and knocking together the larger blocks reduce them to boulders and pebbles, and at last to sand and mud. As many a shore shows, the conglomerate itself is sooner or later subject to the like processes and its cemented masses of heterogeneous component lying on the beach are broken up, and worn away by impact and attrition." And so the compound we call *mind* is slowly reduced to some primitive elements by the stress of age and the storms of time, and I shall endeavour to point out some of the more common signs of such dissolving.

Dissolution may be the natural end of the worn out mind and may be uniform, and like the famous post-chaise of Wendel Holmes all dissolving simultaneously, but this or anything like this is rare; the mind breaks down in different directions according to the nature, education, and surroundings of the individual.

There is no one sign which can be taken as a certain indicator that mind is dissolving, though probably memory will be found to be the best test of decay.

In dissolution, though all the symptoms are evidences of loss of power, this shows itself in different ways, and I prefer to consider separately those cases in which there is defective harmony in the working of the parts of mind, those cases in which there is defect of power of control, and those in which the decay is marked by real evident loss of power or faculty.

There is defective power of acquisition.

Defective power of co-ordination.

Defective powers of control.

Or to put it another way there may be loss of constructive power, loss of memory, loss of control of volition, and failure of

judgment, and one or more of these defects may be more marked than the rest.

By mental dissolution I mean the slow breaking down of the mental organisation, such as is best seen in senile states, but which may arise under other conditions. This dissolution may be looked upon as directly related to waste or disease of the higher nervous tissues; it is associated with decay, in fact, it is best seen in *natural*, *i.e.*, in *senile* decay, but is very readily traced in premature brain decay, such as is met with in the so-called general paralysis, and in some toxic states, such as in alcoholism. Decay of brain, like the ruin of buildings, may proceed at very varying rates; the rapidity depending chiefly on the nature of the structure attacked and on the physical changes which cause the decay. Just as in a building the rubble-built house falls before the stone one, so the brain injured by or badly built under the influence of alcohol falls more easily into ruin than that developed under healthy natural conditions; the brain of the city liver tends to break down prematurely more readily than the brain of the peasant.

The nature of the surroundings also affects the ruin of the house; the climate of Egypt allows ages to pass over monuments, leaving but little trace of the passage of time, while the storms of our climate cause rapid disintegration, and so with mind, a passionless existence leads less readily to dissolution than does the life of action. I shall point out that varying conditions lead to the same results as age.

In dissolution we notice that there is a reverse process to that of evolution, but this statement does not cover all that we shall have to notice, for the structure of the mind is complex, and the elements do not decay in equal proportion.

As a rule, we may say that the process of dissolution passes from the higher to the lower, *i.e.*, reducing the more complex and the latest developments to the less complex and more firmly established. In considering dissolution I shall point out that the various so-called faculties of mind suffer differently, and that there is defect of *control* as well as loss of *power*, and much of the mental disorder results from this defect of control.

As it will be quite out of my power in the short time at my disposal to give a full analysis of the subject, I shall endeavour to take a few of the more prominent points.

Before proceeding to more detail, I will take as a preliminary study the marks in general paralysis which indicate the processes of dissolution. First, then, there is almost always emotional instability, what may be called exaggerated emotional reflex, the higher control is lessened ; this may be shown in simple emotion, such as tears, laughter, hysterical displays and the like, or it may be marked by uncontrolled desire (lust) of various kinds. Next there is often exaggerated self-consciousness, which may lead either to hypochondriacal feelings or to exaltation of ideas. Sympathy may assume an exaggerated and emotional aspect ; loss of power may be shown by defective recent memory, by want of judgment or want of energy or power to decide ; further, defect of power may be shown by real weakness of mind assuming the aspect of dementia.

In the general paralytic, as a rule, the special acquirements fail before the more general ones, the musician failing in his most special powers ; thus I have known a man following accurately the notes of music, though he had lost all power of expression, and the artist, though for a time able to draw, yet very deficient in appreciating colour relationships. The accountant may be able to correct another's errors while he makes more grievous mistakes himself. Not only are the defects produced by dissolution to be looked for in the highest, but in the lower, nervous relationships, so that there may be defect of control leading to defect of power over the muscles, as shown in the various modes of expression by look, speech, and writing.

One point to which I shall refer later is that there may appear in some instances to be an increase of power depending either on loss of power of control or loss of sensibility. In the one case there is a wasteful expenditure of capital ; in the other there is a disregard of the former indications of waste, such as painful working.

To take then some of the symptoms in the order I have named : the defect in power of acquiring new groups of facts, such as languages, arts or the like, marks the full tide, if I may use the term, of mind, and is, I believe, generally the first sign of age or decay. I presume many of my hearers fully appreciate this loss. The power of acquiring fresh knowledge varies greatly even in decay and in age ; thus some men have learnt new languages (even the more difficult ones such as Greek) when long past



middle life. I believe the defect of further growth-power need cause no busy man trouble, but it is at the same time an indication of the turning point in mental growth which should not be neglected. This defect is allied to loss of recent memory, to which I shall refer again in more detail. Organically, it seems that there comes a point when the reaction to surrounding impressions is slow and more difficult to make, and a point may be reached where there may be complete inability to retain impressions for a sufficient time to allow a permanent record to be formed.

Dissolution may be heralded by emotional motor disorders besides defects of movements of various kinds; thus, I have met with several well-marked cases of hysterical attacks in old persons of both sexes, and with the hysteria there may occur the same moral perversions which characterise that disorder in young persons; the occurrence of this senile hysteria does not seem to me to be of very grave moment except as an indicator that senile changes are at hand. I do not think it can be taken as a warning that hemiplegia is impending. It is of much more grave import in patients suspected to be general paralytics. It is a serious symptom if the convulsive seizures have an epileptic character, *i.e.*, if they affect special parts of the body, are followed by unconsciousness and sleep, if they have, in fact, a truly epileptic aspect; such fits are of serious import, but they may recur at irregular intervals for some years, and if the patient take care to avoid over-work, over-fatigue, and is careful as to diet, he may be but little worse for an occasional fit of this kind. In some cases these senile epileptics still go on doing their work, though they have to be more restricted in the expenditure of their energies. I do not think that bromides do much good for these cases; mental decay depends on the number rather than the severity of these attacks.

With senile mental changes it is not uncommon to meet with further loss of higher control as evidenced by maniacal excitement, and there are several peculiarities in this excitement which need attention. In some cases very slight physical or moral causes are sufficient to produce violent outbreaks of temper which may pass into a delirious state which generally ends fatally. These cases of senile delirious mania are examples of an acute disease developing on chronic changes, and the prognosis depends

on the active destructive process following long-standing degeneration. In these cases, feeding and rest are the only means likely to do any good, and it must be remembered that stimulants are essential and must be given with no niggard hand. It is much more common to meet with cases in which the loss of control is progressive. Thus, an old man becomes more and more irritable, is more inclined to resent any supposed slight, he loses control so that when he gets angry he becomes beside himself with rage. Such attacks recur and may lead to an almost constant state of rebellion against control; in some cases these outbreaks never become more than attacks of rage, in others they pass these bounds and have to be looked upon as true maniacal attacks. Sometimes after these attacks, which are generally of short duration, there may be periods of calm or even depression, and these periods of depression may be again followed by storms of mania: the mania is almost always associated with violent impulsive acts which, but for the physical weakness of the patient, would more often lead to casualties.

Many cases of this kind pass through many recurrences of this nature, some being left permanently weak-minded, while others return to health, though the mental standard has been somewhat lowered by the illness.

With senile changes, probably melancholic states of mind are the more common; these divide themselves into the various forms of mental depression, some being chiefly hypochondriacal, while others are more allied to ordinary melancholia, and may be either active or passive in their expression. Many old patients have recurring attacks of mental depression, and I am inclined to think that this form of senile melancholia is as easily transmitted from parent to child as is the gout. I meet with cases very frequently in which there is a history of mental depression running through the family in all its old members. It is important to remember that members of such families are often suicidal and not to be trusted, but in my experience that is rather the result of whether some ancestor killed himself, for otherwise simple melancholia without suicidal expression may repeat itself through several generations.

I know of several families of distinguished men, both in politics and in the professions, in which the shadow of old age has always a deeper shade of melancholia attached to it.



The hypochondriacal old man is, as a rule, a long liver and one who gets more and more selfish and exacting as he grows older; he sleeps, badly, but, I believe, not so badly as he thinks.

Suicidal symptoms may arise during any stage of decay; it is not uncommon as a result of the feeling of loss of capacity and the dread that it may cause loss of situation or of position; it may be looked upon as a variety of exaggerated self-feeling hypochondriasis, if you will.

The general cause of suicide in old men is a dread of ruin and a fear of the workhouse. This may occur in women, and recently I saw a French governess who killed herself by most determined wounds of the abdomen because she thought all her money was lost. Some old men are very determined in their attempts, some being gouty, and I have met with this as a special variety, as in the case of a captain in the Royal Navy.

A very practical question arises as to which senile cases are to be treated as suicidal: I reply, no senile melancholiac can be trusted; but there is but little danger from persons with much loss of memory—such persons may impulsively injure themselves or others, but they have no persistent idea. I am in the habit of saying that the danger of suicide is very slight in cases where recent memory is wanting.

#### DISORDERS OF MEMORY IN OLD AGE.

The most common symptom of dissolution is defect of recent memory; this varies greatly in degree, ranging from the tendency to narrate tales of the past to the glory of the teller to total forgetfulness of every recent impression.

The loss is rarely as complete in these cases as it is in cases of alcoholism, though I have met with patients who having had a meal straightway forgot this and ask for another almost directly. There is frequently a recollection of isolated facts in these senile cases, so that, though the patient forgets the address of the house he is occupying, or the day of the week, yet he may recall the fact that he has told the doctor never to cross his door again, or he may remember that he discharged his valet, and be indignant at his reappearance.

The memory of the past may be also confused, so that, though it appears to be good up to a certain point, those who know the life



history recognise gaps even in the part which seemed to the outsider to be perfect. In some cases there are what I have called denudations, that is, things which if not forgotten, have been buried, are revived with all the vividness of recent impressions, and may be believed in as if they were recent events.

Another variety of disorder is that in which there is great confusion as to time and place; this resembles what is often met with among alcoholics, who will freely describe things which they say they have done and places which they have visited, though they have not moved a step from their rooms. A variety of this occurred to me recently in a man of over 80, who thought that all things were done more than once; he thought the clocks were all put back to deceive him, and that the train stopped more than once at each station. He had a kind of mental double vision, and my interpretation of it is that he has two impressions, one in each hemisphere, and that—as in dreams—there is no power of judging time or distance from pure sensations; these are referred to as distant impressions; anyway the fact remains that in some cases of senile dissolution one meets with such mental double vision.

I should like to hear the opinion of Fellows as to whether with the loss or defect of recent memory there may be a real increase—revival, if you like—of distant memory. I have been much astonished by listening to, and afterwards investigating, the stories of some old men: the vividness of their stories and the minute detail has made me wonder if it were possible that there should be revealing denudation, to use my own term.

I have elsewhere referred to some of the organised disorders of the emotions, hysteria, which may occur with senility. There may be very marked loss of control of the emotions, but it is interesting to note that with this there is often a marked loss of real feeling. I think this is of importance, and should be looked at carefully in its several relationships. First then, the old person may be emotional, may rage at nothing, and may, and generally does, cry over trifles, but the deeper griefs do not stir him, or perhaps it would be better to say that what would be crushing weights to younger persons cause but a momentary sorrow; I have known an aged mother lose her only son, to whom she had been deeply attached, and yet on the news of his death shed a tear and then resumed the reading of the novel she was at. It has been thought that with age a stage of philosophical calm has come, but

I believe the calm is one of organic, not philosophical, origin. The wind is tempered to the shorn lamb.

We know that with age there is hardening of tissue, a tendency to rigidity, and with this the special senses fail, but I want to know why so many old men, like so many degenerating general paralytics, develop great appetites and increased power of digestion.

Many a man who has been a martyr to dyspepsia for years reaches an age, as a rule the wrong side of 60, when he can eat and drink with impunity things which had for years disagreed with him; for example, I have known men who for years could take neither beer, tea, nor coffee, and to whom cheese was a deadly food, yet later could enjoy each or all of these things.

Shakespeare represents Lear as calling loudly for his dinner, "Let me not stay a jot for dinner," and I have no doubt he had noticed the old man's appetite. I could more readily understand the increased appetite due to loss of feeling of satiety, but that the digestion should improve and that there should be gain in weight is rather surprising. We know that general paralytics gain weight for a time, the weight being due to a production of a lower tissue fat.

Allied to the question of loss of emotional control we cannot neglect the development of eroticism. This occurs in both sexes, but in my experience the redevelopment of lust is more common in women at the climacteric, and in men with senility. What it is due to is doubtful; I have no doubt that in some cases it is due to local prostatic irritation, and I have been able to verify this in some cases, but in others I could detect no local cause, the whole seeming part of a dissolution which took place on the same lines as development, and men on the road to second childhood passed through a period of second youth. This is confirmed by the hysterical attacks which may then occur.

There are unfortunately many examples of sexual perversion in old men, and I always read with pity the tales of old men who are convicted of indecent assaults and the like; many a man's reputation is shattered in consequence of arterial degeneration and decay of brain. I have known great moral leaders thus ruin their fame. In one case an old man lost his higher inhibitor, in the shape of a good wife, only to sink into lust and disgrace.

In rare cases the sexual instinct persists to extreme age: I have



known it to be present over 90. This manifestation need not necessarily be pathological, but it must be so considered when the sexual passion has been dead for some years and then suddenly revives.

The abnormal manifestations of sexual life may be the precursor of senile dementia, and may make itself clear before any intellectual change is discovered. Careful examination will, however, detect change in the tone of mind of the man.

The evidences in such cases are lascivious speech (a kind of Rabelais stage), then gesture. Generally the next danger arises from children. This noxious selection of weak victims is very characteristic. Defective sexual power and greatly diminished moral sense explain the additional fact of the perversity of the sexual acts of these aged men. Neurotic delirium and satyriasis may occur.

There may be complete sexual perversion with attraction only for members of the same sex. I have met with this both in men and women. Another group which one constantly meets with is that of the senile reformers. At all ages, one meets with the sexual sinners who wish to become reformers, one meets with the impotent masturbator, the celibate, and the old man, each willing to do good to those who have fallen, and in most instances the reform does not begin at home. Beware of this tendency in men, whether threatening with general paralysis or age.

Many old men become misers—there is a tendency to collect which is eminently associated with age, and besides this there is a variety of collectors who, without having the miser's instinct for collecting, really are governed by the dread of poverty and ruin, which is one of the common phases of old men's melancholia. One old doctor patient of mine passed nothing in the street which could be collected: he had hundreds of sardine tins, horse hairs arranged according to colour and length, pins and nails, all carefully put away for future use; he too was more careful of the *material* than the *moral* good. With collecting very commonly goes a disregard of the duties of cleanliness, and I have never been quite able to understand the reason for this. It is not indolence always, for some dirty persons have been restless and always on the move, nor is it the accentuation of a habit, for only recently I saw an old aristocrat whose whole life had been that of a wealthy refined Englishman, a man who for three quarters of a century



never omitted his bath, yet now, at nearly 90, has to be kept clean by force, though beyond loss of memory there is little to note his senility. Many have been the praisers of old age, and with it what we may call dissolution there is pointed out to be a smoothing of our way to the tomb, a detaching us from the present world; all this may be true enough, but my own experience is that the feeling of a loosening hold on the world as we get older is not a fact.

The PRESIDENT mentioned two cases which illustrated a point brought out in the paper. One was that of a well-known man, who, in the full zenith of his intellect, while stopping in a provincial town, had a standing invitation to luncheon at the house of a friend. One day he went in and lunched, and an hour later returned and asked for lunch again, being apparently unaware that he had already had a meal. In another case, a gentleman, who had in the morning made all the arrangements for a *conversazione* and supper party in his house for the same evening, came home and went to bed, having forgotten all about it.

Dr. PASTEUR referred to a class of cases occasionally met with in which persons of 50 or thereabouts were seized with general epileptiform convulsions. Usually there had been no previous attacks, and prodromata were often wanting. In two or three cases of this kind which had come under his notice the convulsions had been very severe. In two the patient had passed into the status epilepticus, with high temperature and albuminous urine. In the course of two or three days, however, these patients became restored to their usual health, and every objective sign of disease had disappeared. He would like to ask Dr. Savage whether these convulsive attacks were indicative of commencing mental dissolution.

Dr. FRANCIS WARNER remarked that he had often seen dissolution of all mental impression on the faces of children and adults whose faces were in no way paralysed, as to coarse movements; such is common in paralysis agitans. On the other hand children whose faces are frequently distorted with grimaces or athetoid movements may at the same time retain those fine shades of muscular action which, being unable to describe anatomically, we commonly term "mental expression." He enquired if Dr. Savage thought that cases of defective development were more prone than others to mental dissolution and nerve disturbance; his own observation appeared to indicate that this was the case.

Mr. J. PEEKE RICHARDS was of opinion that the inordinate appetite referred to by Dr. Savage, as so often occurring in cases of senile insanity, was not due to the mental condition of the patient, but was attributable to the pathological condition of the mucous membrane of the stomach so often found in persons of advanced age, the sentient extremities of the nerves in the mucous lining of the organ having become blunted by the constant wear and tear of many years' digestion, thus enabling individuals in whom this condition was present to digest easily what in previous years would have caused them much pain and suffering. He also thought that the loss of memory as to recent events, considered by Dr. Savage as an evidence of mental deterioration caused by advancing years, was due in many instances to absent-mindedness, such as is often seen even in young persons, and was not caused by senile changes in the intellectual centres.

Dr. SAVAGE, in reply to Dr. Bristowe, said that he recognised that there might be lapses in memory such as described by him, without there being any ground for supposing that there was any dissolution of mind going on. Such instances were not infrequent, and the individuals were only considered as eccentric. Dr. Pasteur had referred to cases of epilepsy which occur in patients between 50 and 60 ; these belong to the group to which Dr. Savage referred, and he considered such cases had not yet been fully recognised, at least in England. In reply to Dr. Warner, he thought that there was no doubt that those minds which were the more highly trained were more liable to show signs of dissolution, and that this decay was more common among the more highly educated than among the illiterate. It is quite certain that the higher the structure, whether mental or physical, the more conspicuous it is in ruin. A tower of 100 feet would probably decay more readily than one of 10 feet, built of the same material, and certainly its decay would be more evident. Dr. Richards had remarked on Dr. Savage not noticing more in detail the restless and troublesome cases of senile dementia. His reply was that the object of the paper was rather to point out the more marked symptoms which were met with outside asylums. He fully recognised the cases of restless disorder, and knew them to be very troublesome to deal with.

## CONSTITUTIONAL DIFFERENCES BETWEEN BOYS AND GIRLS, AND THEIR RELATION TO EDUCATIONAL REQUIREMENTS.

By FRANCIS WARNER, M.D. Lond., F.R.C.P.

IN bringing before you some account of the constitutional differences observable in the bodily and brain conditions of school children, I propose to base my remarks on a Report\* of 50,027 children (boys 26,884, girls 23,143) whom I have seen individually in 106 schools, and regret that I have not here found space for quotations from kindred investigations and Hospital Reports. In this investigation I saw the children individually upon a uniform plan, and took notes of all cases in some point below the normal (boys 5,579, girls 3,607), and the teacher's report concerning each of these cases was recorded. It must be remembered that my tables refer mainly to the 20 per cent. of boys and 15 per cent. of girls who are in some point below the normal. The normal child is well made, and his tissues are well nourished ; the environment, past and present, has well co-ordinated his bodily growth and the balance of his nerve centres.

\* Presented to a joint committee of the British Medical Association and the Charity Organisation Society.



For an account of the points observed, I must refer you to the Report published, but it will be seen at once from the statement made that there is a great difference observable between the sexes—more deviation from the normal among the boys, except for “low nutrition,” a point with which I shall deal presently.

The cases noted fall naturally into four primary groups.

- I. “Development cases,” where there is some defect in size, form, or proportioning of the body or its parts; this is the largest group.
- II. “Nerve cases,” where movements and attitudes seen in the body indicate defect or disorder in the action or balance of the nerve centres or their reactions through the senses.
- III. “Nutrition cases”—children thin, pale, or delicate.
- IV. “Dull children”—the pupils reported by the teachers as below average ability in their lessons.

How do these boys and girls respectively bear the effects of their environment? The reply may in part be sought among the “nerve cases,” the “nutrition cases,” the “dull pupils.” In all groups of schools, as tabulated, it is the “development cases” that are most numerous and wear the worst; it is abnormality in make that is accompanied by low power of resistance in nerve and nutrition, and that is largely accompanied by mental dulness, the nutrition and - dulness in these cases standing in large percentage against the girls.

“Nerve cases” include cases of defect and disorder of the nerve system; many of the latter are due to want of the training necessary to the acquired normal co-ordination of the nerve centres. Boys form the larger proportion, but if we deduct the large number of cases of “frontal muscles overacting,” the proportion of nerve cases becomes about 7 per cent. upon the 50,000 for boys and for girls; this sign appears largely associated either with physical defect or want of proper training.

“Children pale, thin, or delicate.” This group is more numerous among girls; it is very largely associated with defect in development; if we deduct the cases in which it is associated with development defect, the proportion of boys and girls becomes equal, viz., 1 per cent. for each.

The worst groups of children seen were as follows:—



	Boys.	Girls.
Children exceptional in mental status, including idiots, imbeciles, and "children feebly gifted mentally" .....	124	110
Epileptics and children with history of fits during school life .....	36	18
Crippled, maimed, paralysed, deformed .....	155	84
Children of low constitutional power and dull, <i>i.e.</i> , cases appearing in each of the four primary groups .....	192	157

Making allowance for cases appearing in more than one of the groups above, we have 473 boys, 344 girls. These children, it is thought, should be known to school managers as probably requiring special care and training, and their cases should be considered separately; the proportion upon the school population is for boys 17 per 1,000, for girls 15 per 1,000.

"Eye cases," boys 836, girls 637, include boys 485 and girls 322 with squint or varying strabismus.

Having described the four primary groups of children, as formed by observation and tabulation, we may try and look upon the children from the school point of view. The child is sent to school and must be dealt with by the teachers; from what I am told it appears to take from three to six weeks before the teachers can form an opinion as to the child's capacity. The School Board require that a child should be kept in school for six months before it can be presented as requiring special instruction on account of its feebleness; inspection enables the points seen in the child to be summed up in a few minutes. Gradually, by unconscious observation, the teachers become aware in a child that there is low nutrition—brain disorder or mental dulness—each factor may require separate treatment. In fact, the training and management of a child needs to be adapted to the combination of conditions present, by which alone we have about 250 groups of children. The relative effect of each factor in the defectiveness differs in the sexes; hence I conclude that different methods are needed in the management of boys and girls. Take 100 dull boys and 100 dull girls: of the boys 18 are of "low nutrition," but of the girls 27—the average condition of dull boys and girls differs. Among the normally made children there are more dull boys, and "low nutri-

tion" is equally distributed in the sexes; among "development cases" there are more dull girls, and more thin girls. The teacher may see an awkward twitch, tricks of the hands, eyes inattentive not fixing well, head lolling, spine bent (lordosis), &c., or other "nerve sign." Now, taken as a group, "nerve cases" are more common with boys than girls; taking 100 "nerve cases boys" and 100 "nerve cases girls," of the boys 18 are of "low nutrition," 40 are dull; of the girls 29 are of "low nutrition" and 42 dull—in both particulars the girls are in worse condition. There is a large proportion of abnormally made boys, but the condition when present tells more against the girls. Let us take 100 "development cases boys" and 100 "development cases girls" and see, according to experience gained by observation, how it fares with them in day schools and in resident institutions. In the latter the number of thin or pale children falls for boys from 23 to 16, for girls from 38 to 20; "nerve cases" increase from 50 for boys to 62, and for girls from 47 to 52; dull cases increase from 38 for boys to 40, and for girls from 40 to 44. Institution life is specially ill-adapted to boys, and apparently not well adapted to either sex. We may group children as to social class in elementary day schools with similar standards under Government inspection. Each of the four primary groups is found more frequent in the upper class, but the incidence upon the sexes varies. As to girls there is an equal percentage of "development cases" in the upper and poorer classes, but for boys this is 14 in upper class and 11 in the poorer; I think the fact of equality of development cases among the girls removes the argument that the upper class have more defects from survival of weak children who die among the poor.

Taking the "development cases," the 100 boys in the upper class present 48 with "nerve signs," the girls 54; for a similar group in the poorer schools this would be 51 boys and 45 girls. Whatever be the difference in the classes the advantage is thus on the boys' side. I suspect it is increased hard work which suits them better than the girls.

Lastly, we will take schools arranged as to nationality, and comparing the English, Irish, and Jews: the Jewish children come out as the best, the English rather lower, and the Irish, especially the boys, presented the largest proportion of noteworthy cases.

I am unwilling to generalise upon insufficient data, but the



subject before us appears both interesting and important, and a temporary hypothesis may aid future observations. The great characteristic of young living things, including children, is the tendency to spontaneous growth and action in cellular tissues—including the brain, not much controlled by external impressions in the earliest stages. The more uniform the environment has been in antecedent generations the more regular will conformation of body and mode of brain action be, as among the Jews. I think it probable that the irregularities termed “defect in development” are due to spontaneous growth, the malproportioned parts not being duly under the control of past environment, while the tendency to abnormal nerve signs is likewise largely due to spontaneity among the nerve centres. Retentiveness is antithetical to spontaneity; spontaneity leads to abnormality if there be not enough retentiveness of inherited impressions. There seems to be more spontaneity in boys, and when the weary labours of training them have been brought to a favourable result they present greater power than the girls.

See how the facts given bear upon the hypothesis. The “development cases boys” present the greatest spontaneity in growth, and shut up in institutions the brains remain unimpressed by the varied environment of the streets; they remain full of spontaneous movements and ill-balanced nerve actions. In the most ancient nation growth and brain action are most uniform. In the infant school constant spontaneous movement is the characteristic of healthy brains; this I described under the term “microkinesis,” and at this stage all postures (except a few seen in athetosis, &c.) are normal. Many of the abnormal nerve signs are, I believe, reversions to this spontaneity of infancy; dissolutions of acquired co-ordination. Finger twitching is more common in boys than girls: chorea is accompanied by fall of body weight and is more common in girls. Many neurotic states are accompanied by fall of body weight, and I expect that the low nutrition to which delicate girls are shown to be so prone under certain conditions tends to fix nerve disturbance more with them than with boys. Hysteria may be thought to be an exception, as often the body remains fat, but the cure is in brain stimulation by interesting occupation, which aids brain nutrition. It appears that in conditions of low nutrition the brain is more spontaneous in its motor action, and less under due control by influences impressing it—this is illustrated in chorea.



Any attempt to remove the frequency of defective development must be made by the efforts of State medicine; as work in this direction, I have shown that as far as these observations go the distribution in different localities is unequal. The small head is obviously a common girl defect, but it seems most common among large block dwellings and warehouses.

The nerve defects can largely be dealt with by educational training, but they are at present not commonly known to teachers, who can, therefore, make no systematic effort for their removal. As to any differences that may be recommended between the care of boys and girls in school, it must be remembered that school arrangements are made for the average or normal children. It seems desirable that teachers should themselves have some instruction on the scientific observation of children, and that at least those children who appear weak, nervous, or dull should be seen by some one competent to advise. Though there are fewer girls of constitution below the normal, still they may fall into ill health from conditions which produce less harm in boys, and care adapted to their needs may enable their education to proceed. I think the evidence collected points to the great advantages of the national education in producing a normal and a stable brain state, aiding evolution to a uniform type. Improvements are undoubtedly needed,\* and physical training should be further adapted to produce well co-ordinated nerve action as well as muscular development. There also appears to be many ways in which the strength of both pupils and teachers might be economised without diminishing the amount of instruction given. If those in charge of children knew more of their bodily and brain condition many educational problems might be simplified, and the needs of girls in particular might be better met.

\* See author's work on "Mental Faculty." Cambridge University Press.

Classes and sub-classes of cases as distributed among 50,000 children.	Percentage of cases in numbers seen.		Number of cases.	
	Boys.	Girls.	Boys.	Girls.
<i>Among the 26,884 boys and 23,143 girls seen—</i>				
Of the 50,000 cases seen, the proportion noted				
the "development cases" .....	20.0	15.0	5,579	3,607
" " the "development cases" .....	13.4	9.6	3,616	2,235
" " the "nerve cases" .....	12.6	8.9	3,413	2,074
" " the "nutrition cases" .....	3.8	4.2	1,030	973
" " the "dull cases" .....	8.2	6.3	2,216	1,463
Of the "development cases" proportion with "nerve signs" ...	54.6	49.0	1,975	1,096
" " " " "low nutrition" ..	20.2	32.0	733	726
" " " " "mental dulness" ..	38.3	41.5	1,398	928
Of the "nerve cases" proportion with "development defects" ..	57.8	52.8	1,975	1,096
" " " " "low nutrition" .....	18.6	28.8	635	598
" " " " "mental dulness" .....	40.1	42.4	1,370	880
Of the "low nutrition cases" proportion with "development defects" ..	71.0	74.6	733	726
" " " " "nerve signs" ..	61.6	61.4	635	598
Of the "low nutrition cases" proportion with "nerve signs" ..	39.0	40.5	402	395
" " " " "mental dulness" ..				
Of the "dull cases" proportion with "development defects" ..	63.0	63.0	1,398	928
" " " " "nerve signs" .....	61.8	60.0	1,370	880
" " " " "low nutrition" .....	18.0	27.0	432	395

Showing in groups of schools the distribution of classes of cases.	Percentage of cases.		Number of cases.	
	Boys.	Girls.	Boys.	Girls.
<i>In Resident Institutions</i> : 8,246 boys ; 5,403 girls— “Development cases,” proportion with “nerve signs” . . . . . (boys, 1,324 ; girls, 671) proportion with “low nutrition” . . . . . “ ” “mental dulness” . . . . .	62 16 40	52 20 44	820 199 528	353 133 297
<i>In Day Schools</i> : 18,638 boys ; 17,740 girls— “Development cases,” proportion with “nerve signs” . . . . . (boys, 2,292 ; girls, 1,564) proportion with “low nutrition” . . . . . “ ” “mental dulness” . . . . .	50 23 38	47 38 40	1,155 534 870	743 593 631
<i>In Day Schools of Upper Class</i> : * 5,281 boys ; 4,934 girls— Of the numbers seen, proportion of “development cases” . . . . . “ ” “nerve cases” . . . . . “ ” “nutrition low” . . . . . “ ” “dull cases” . . . . . Of the “development cases,” proportion with “nerve signs” . . .	14 12.7 4.8 8.7 48	8 10 5.6 5.8 54	756 — — — 370	419 — — — 228
<i>In Poorer Day Schools</i> : 13,357 boys ; 12,806 girls— Of the numbers seen, proportion of “development cases” . . . . . “ ” “nerve cases” . . . . . “ ” “nutrition low” . . . . . “ ” “dull cases” . . . . . Of the “development cases,” proportion with “nerve signs” . .	11 10 3 7 51	8 7 4 5 45	1,536 — — — 785	1,145 — — — 515

\* Public elementary schools for middle classes.



Dr. NORMAN KERR held that our present system of education was wrongly organised. The Jews were examples of the reward that followed strict obedience to the laws of health in body and mind. He looked upon malnutrition as a great cause of mental as well as bodily defect. As much muscular exercise as possible should be co-ordinated with mental training.

Dr. SAVAGE referred to the amount of work which had been done by Dr. Warner, and, as a member of several committees on which Dr. Warner had also served, Dr. Savage could say the work was almost all done by one man, and that man was Warner. The tables were full and interesting, and the importance of the subject was not yet understood, or it would be a national question, not a parochial one. The system of Dr. Warner was intended to detect the incipient criminal and ne'er-do-weel, and stop him in his downward course, and by special education turn him to a useful career. The sieve which Dr. Warner was making was to strain from the ordinary school population the dangerous and imperfect members, so that they may have particular and special training. There may be doubts as to how far this may be carried, but no one could judge till he had studied the method of Dr. Warner. There are certain parts of the tables which may be fairly criticised; thus to speak of the "signs" among the Irish is misleading, for it really is only the Irish of cities which somehow seem to be the dregs of the nation. There are no tables to show what the state of the Irish is in Ireland. Dr. Savage said that in years gone by he had been interested in the subject of the absence of certain forms of degeneration among the Irish and other Celts, but he soon found this absence was only to be found in the simpler homes and country lives of the Celts, and that these persons, when they moved to cities, were at least as liable to the degenerations as others. The tables in reference to the Irish and the Jews must therefore be only accepted as referring to them when found in cities.

Dr. ALTHAUS regretted that time would not allow of a full discussion of Dr. Warner's paper, which had evidently been prepared with great care and industry. He would, however, take exception to his statement that Jews were less neurotic than Christians, as he had for many years past found a much larger proportion of neurosis amongst Jews than amongst Christians; and this was also the experience of Professor Charcot, with whom he had lately discussed this point. He attributed the greater liability of Jews to neuroses to the persecution which that race had undergone for so many centuries.

Dr. SOLOMON SMITH asked whether Dr. Warner had extended his enquiries to departures from the normal in the direction of precocious brilliancy as well as in that of dulness. It was a matter of considerable interest to know whether the cases of precocious brilliancy in early life, a few of which existed in every school, and which often led to little later on, were to be looked upon as higher types, or whether they were connected with the same developmental and nutritional defects as Dr. Warner had shown to be such common accompaniments of dulness.

Dr. WARNER remarked that he did not think there was any great amount of starvation, as the term is commonly used by medical men, among the children he had seen. Development defect was largely found among children, "pale, thin, or delicate"; deducting these cases, only 1 per cent. of boys and of girls presented low nutrition. He thought the large amount of defectiveness among the children a matter urgently calling for attention as a department of State medicine, particularly as

to the effects of the buildings. He explained that the Jewish children were seen in the Free Schools, Whitechapel, and the Irish children largely in poor low schools near London. The Jew boys presented more nerve disorder than English boys.

---

*March 27th, 1893.*

## ON A CASE OF APHASIA FROM A FALL ON TO THE LEFT SIDE OF THE HEAD.

By C. E. BEEVOR, M.D.

GEORGE C——, aged 50, carpenter, came to my out-patients at the Great Northern Central Hospital on November 17th, 1891, suffering from aphasia. From his wife it was ascertained that he had had no previous illness of importance, and of his family history one brother had died insane in Colney Hatch Asylum. He has drunk fairly, but not to great excess. No history of syphilis.

From the account kindly sent by Mr. Down, a medical man who saw him, the patient, who was in his usual health, on November 13th, fell 14 feet. He was picked up unconscious, and remained so for fifteen minutes. Soon after he insisted on walking home, from Finsbury Park to Hornsey, and on reaching his house he complained of pain in the head, and was very talkative, and kept repeating the sentence "Had a fall."

The next day he was drowsy, and speech was much worse, and he could not be got to answer questions; he was seen by the doctor in the evening, and only said "Yes! Yes!" in answer to questions, and occasionally made remarks on his own account, but the words easily became slurred and unintelligible. There had been no vomiting; pupils were equally dilated and reacted sluggishly to light. The left side of his face did not seem to move quite so well as the right; there was no sign of paralysis in the limbs. Although he could not answer, he seemed to understand to a certain extent what was said to him, putting out his tongue when told to do so. A week later he was again seen by the doctor, when he seemed not to comprehend anything said to him, nor was he able to explain what he wanted.



Patient came alone to my out-patients on November 17th, four days after the accident, and before any history of the occurrence was obtained, and it was then noticed that he had no definite paralysis; he talked fluently, but it was nonsense, and every now and then such automatic sentences as "Thank you," "This way," would be uttered. On November 19th he had a fit, in which his wife says he made a dreadful noise, was convulsed, and lost his senses, but did not bite his tongue.

On the next day he again came to the hospital and was examined more thoroughly. A healed wound was found about 2 inches above the left external auditory meatus. On examining him he appeared to hear and see quite well, and no hemianopia could be made out.

The speech was affected in the following way:—

He could speak voluntarily, but it was for the most part nonsense, with every now and then intelligible sentences put in automatically. With regard to commands and questions asked verbally, he could perform simple actions, such as putting out the tongue, showing the teeth, standing up and sitting down. When asked what was his name, said "Norbin, Easbin." "How old are you?" replied "Norbin, nothing, no other wheel." Asked how long ago was the accident, said, "Two weens, two ones, no meat, moden through." He, therefore, could understand sufficiently to perform simple actions, but was unable to give his name or his age, and apparently did not recognise his name when it was mentioned to him. He could not understand commands printed or written down, when shown the words "Stand up," he said the words "Tubbun, five pun, one, five pun, four pun"; asked what it meant, he said he did not know.

When told to write anything, such as his name, he wrote fairly well "Chanartian," which was probably an attempt at Chamberlain, when given A to copy he wrote "Charant." He could not repeat any word or letter named to him. He could not pick out objects and letters named to him. He could not write from dictation. He was quite unable to name any object or letters. The patient was admitted into the hospital on November 20th, 1891, under Dr. Burnet, who has kindly allowed me to use the notes of his case.

While in the hospital patient improved so that on November 22nd he was able to recognise the letter A when shown to



him and to pronounce it. On November 24th he knew his name when it was written down, and was able to pronounce it. He is now able to copy printed to printed characters. When shown the printed words STAND UP, was able to understand the command, and was able to do it.

On November 27th he could copy printed to writing characters; he could write from dictation, and was able to write spontaneously a letter consisting of a few words, in which there were not many mistakes.

On December 2nd he could read a newspaper and understand it, and could do simple addition sums.

Patient left the hospital, and on December 8th I saw him again as an out-patient. He talked very well, but at times used wrong words, such as "impaired" in place of "improved," and spoke slowly, and had to think about his words. He remembered quite well being asked about a watch and a two-shilling piece on his first attendance. On being shown a table he could name it, but did not remember for some time that it was made of ash (he is a carpenter). Shown a piece of ebony, he said, "What a fool I am," but he knew the name as soon as it was mentioned. Blotting-paper he calls "ink-paper." On December 11th he was better, and knew the name of every common object, but his speech was still hesitating. He continued attending till February 9th, 1892, when he talked quite fluently, and only hesitated for a word here and there.

He has come once or twice since, and appeared again last week for slight bronchitis; he thinks that his memory is not so good as it used to be, and he very easily forgets the names of people and things.

I have brought this case forward as an illustration of sensory aphasia from a fall on to the left side of the head.

In examining cases of aphasia, I have followed the classifications of various writers, but especially that of Dr. Bastian, which he gave in a paper read by him before the Annual Meeting of the British Medical Association in 1887.\* Dr. Bastian there takes the four chief centres connected with speech, viz., the so-called motor centre for the muscular expression of words, which Dr. Bastian terms the glosso-kinæsthetic centre (the difference in the two

\* 'Brit. Med. Jour.,' Oct. 29th, Nov. 5th, 1887.

words involves the discussion of a theory which it would not be possible to go into with the time at my disposal), the auditory word centre connected with this, the visual word centre in connection with the auditory, and lastly the motor or kinæsthetic centre for the power of writing.

These four centres are combined in such a way that the auditory and visual are joined by commissural fibres, and the motor speech centre is connected with the auditory word centre, and the visual is also connected with the kinæsthetic or so-called motor centre for writing.

The auditory word centre is probably situated in the 1st temporo-sphenoidal convolution of the left side, perhaps at the posterior part, the visual word centre in the supra-marginal and angular gyri, the speech centre in the posterior part of the 3rd frontal convolution, and the centre for co-ordinating the movements of writing in the middle third of the ascending frontal and parietal convolutions. It will be seen that there is no direct connection given between the speech centre and the writing centre, as I must agree with Dr. Bastian that the connection between these two is always through the visual or the auditory centres.

In working out a case of aphasia the following questions are asked, the words in brackets give the part of the brain whose integrity is affected when the action cannot be performed, and the question is answered as it refers to the present case:—

1. Can he speak voluntarily (kinæsthetic speech centre)? Yes, imperfectly.
2. Can he understand spoken commands (auditory word centre)? ..... { Not at first;  
yes later.
3. Can he understand printed commands (visual word centre)? ..... No.
4. Can he write spontaneously (kinæsthetic writing centre)? ..... Yes.
5. Can he repeat spoken words (aud.-speech commissure)? No.
6. Can he pick out named objects or letters (aud.-visual commissure)? ..... No.
7. Can he write from dictation (aud.-vis.-writing commissure)? ..... No.
8. Can he copy printing to writing (vis.-hand commissure)? ..... No.
9. Can he name letters or objects seen, or objects heard, felt, smelt, or tasted (vis.-aud.-tactile-gustatory-speech commissure)? ..... No.



10. Can he recognise the use of the object seen, heard, felt, tasted, or smelt (localisation not known) ?..... Yes (?).

It will thus be seen from the history that in the case of the four chief centres the visual word centre was especially affected, and on the second day also the auditory word centre, but that this got better on the third day, so that he could understand simple commands.

The block in the visual word centre would affect all the commissural fibres passing through it, so that, although he could write voluntarily, he could not write from dictation, and although he could speak, he had lost the power of naming objects.

The lesion of the auditory speech commissure is rather difficult to understand, as the auditory word centre apparently recovered after the second day, but there seems no doubt that the impossibility of repeating words was owing to the lesion of the commissure.

With regard to the relation of the localisation of the lesion in the brain to the injury, the scalp wound was exactly 2 inches vertically above the external auditory meatus, and this would correspond to about the middle of the 1st temporo-sphenoidal convolution.

Now the chief lesion, as given by the symptoms, being in the visual word centre, which is considered to be situated in the supra-marginal or angular gyri, it would be a question to decide where to find the lesion, supposing it had been thought advisable to trephine and remove a blood clot.

Against the lesion being in the supra-marginal and angular gyrus is the fact that there was no hemianopia, but this is not an insuperable objection, as some observers think that the hemianopia noticed in these cases is due to the optic radiations of Gratiolet, which come very near to the cortex, being involved, and that no hemianopia exists with a superficial lesion of the supra-marginal and angular gyri. If therefore it had been necessary to operate, I think it would have been advisable to trephine at the scar over the region of the left 1st temporo-sphenoidal convolution, and if nothing were found to work backwards and upwards.

The nature of the lesion was probably meningeal hæmorrhage or simply concussion of the left half of the brain, and the case is interesting from the rapidity with which the man recovered.\*

I would wish to conclude by expressing the opinion that when

\* Since this paper was read the patient has had another epileptic fit, on August 1st, and he has never completely recovered his former mental power.



cases of aphasia do recover it is by reason of the restitution of the damaged left hemisphere, and not by the right hemisphere being educated to take up the functions of the left hemisphere, as I cannot conceive it possible that the place of the most highly organised function of the brain, namely, that of speech, can be taken up by the opposite hemisphere, when it is not possible for the opposite hemisphere to take on the function of such a much lower specialised function than speech, viz., that of the movements of the fingers and thumb. If the left so-called motor cortical centre for the right hand is removed, it is not possible for the right cortical centre to take on the function of the corresponding side and restore voluntary action.

Dr. DE HAVILLAND HALL pointed out that in some cases of aphasia, especially when caused by a blow on the head, anosmia had also been found to be present ; and he asked the reader of the paper whether the sense of smell had been tested in his case. In the event of the anosmia being unilateral, unless the nostrils were carefully tested, the defect would, in all probability, be overlooked, as the patient is hardly likely to notice the absence of the sense of smell in one nostril.

Mr. W. SPENCER WATSON also wished to know whether there had been any loss or disturbance of the sense of smell in this case. The seat of the injury on the skull suggested the possibility of some lesion in the temporo-sphenoidal convolution in which the olfactory ganglionic centre was supposed to lie. It would be interesting to hear from Dr. Beevor whether or not optic neuritis was present in the case related, and whether the recovery was due to treatment or to the spontaneous absorption of effused blood.

Dr. WALTER CARR commented on Dr. Beevor's remarks on the inability of the right hemisphere to discharge the functions of the left in cases of aphasia, and asked if an exception might not happen in the case of very young children.

Dr. BEEVOR, in reply, said that owing to the difficulty the patient had in expressing himself it was impossible to be sure about the absence of smell ; he never complained of any perversion of that sense. There was no change in the optic discs. The recovery, he believed, followed on absorption of effused blood. He did not think that, even in children, the right hemisphere was capable of assuming the functions of the damaged left side.

## CROUPOUS PNEUMONIA IN CHILDREN.

By FRANCIS HAWKINS, M.B.

A YEAR ago, at the request of a practitioner, I saw in consultation a male child aged one year, who was supposed to be suffering from meningitis. The child, small and delicate-looking, had been ailing for one week, and the symptoms, as narrated to me, were vomit-

ing with occasional cough, strabismus, followed or accompanied by frequent movement of the head from side to side, and great restlessness and fretfulness. At our visit the temperature was  $100\cdot2^{\circ}$ ; pulse 120, the respirations being 45. The expansion of the alæ nasi in respiration led me at once to examine the lungs, and on doing so I found dulness at the right base, and tubular breathing with fine crepitation, which was, I thought, due to croupous pneumonia; and I gave it as my opinion that all the varied symptoms complained of could be attributed to the condition of the right lung. For seven days the temperature remained persistently high, being on the seventh day  $103^{\circ}$ , when suddenly, on the eighth day, it fell to normal. The child made a good recovery, and is, I believe, at this present moment, quite well.

Before this, I had seen children *over* five years of age suffering from croupous pneumonia, but never before at so young an age. Shortly after this I saw two cases, one associated with typhoid fever, and another with scarlet fever. Upon these cases and others that I had seen, I resolved, for the sake of personal information, to make a numerical and inductive enquiry as to the occurrence of croupous pneumonia in children, and thought, I trust not wrongly, that this enquiry would be of interest to the Fellows of this Society.

I will now define what I mean by *croupous pneumonia*. It is an acute infective disease, consisting of an inflammation of the pulmonary tissue, in which, from the presence of fibrin and leucocytes within the alveoli and smaller bronchi, the whole or the greater part of one or more lobes is rendered solid. This condition may be accompanied by pleurisy, and, in children, is frequently accompanied by catarrhal pneumonia.

This being my definition, I would ask when, as to month or season of the year, this condition may be most frequently met with?

In answer to this, my numerical investigations of 148\* cases, extending over a period of four years, show that so far as monthly variations in incidence are concerned, most cases occur during March, May, and July, while fewest occur in January, August, and December.

\* To avoid further reference I may here say that nearly the whole of the cases from which I make this investigation are taken from the published reports of the Hospital for Sick Children, Pendlebury, Manchester.

The following are the facts as regards *months of the year* :

Nine	cases occurred in	January.
Thirteen	„	February.
Eighteen	„	March.
Eleven	„	April.
Nineteen	„	May.
Thirteen	„	June.
Fifteen	„	July.
Eight	„	August.
Twelve	„	September.
Ten	„	October.
Twelve	„	November.
Eight	„	December.

It is of interest here to note that of 708 cases,\* mostly in adults, extending over a period of ten years, most occurred during the months of March, May, and September, and fewest during the months of February, August, and December.

As regards the *season of the year*, on examining a similar number of cases, it would seem that the frequency is greatest in spring, while in summer, autumn, and winter the variation is not very great.

Thus 48 occurred in spring.		
36	„	summer.
34	„	autumn.
30	„	winter.

If, now, we compare this with the season of the year of the 708 cases above referred to, we find that, while spring furnishes most cases, autumn ranks next, summer still lower, while winter is lowest in the scale.

Having thus seen that croupous pneumonia may occur at all months and seasons of the year, the next enquiry will have reference to the *previous health and antecedent illnesses*, and their effect, if any, in influencing the onset of this disease. For this purpose, 196 cases in which these particulars were given have been reviewed, and I find that a large proportion of the cases of pneumonia occur quite independently of any antecedent disease,

\* An analysis of 708 cases by Drs. Hadden, Hector Mackenzie, and Wallace Ord, 'St. Thomas's Hospital Reports,' vol. xix.



and that the largest number of antecedent diseases which may have any share in inducing susceptibility are previous attacks of pneumonia and measles. It is possible that a delicate state of health may predispose to it. In 102 cases the previous health had been good, and the patients described as perfectly healthy until the attack of pneumonia. Of the remaining ninety-four cases, twenty-seven were described as always delicate. One had chicken-pox two years before; one had Pott's disease of the vertebræ; three had abscesses in various situations; three had heart disease, two mitral, and one aortic and mitral; one had intermittent fever five years before; two had anæmia one year before; one encephalitis; one inflammation of the bowels; two phthisis; two wasting (cause not stated); one congenital syphilis; one infantile paralysis; one rickets; thirteen measles (but in no case had measles occurred immediately before the pneumonia); three scarlet fever; three typhoid fever; thirteen pneumonia (varying from four months to three years before); one pleurisy; thirteen bronchitis; one whooping cough one year before.

In reading through the records of 220 cases I can only find *seven* instances in which any direct or indirect *cause* was assigned. They are as follows. Chill occurred in one case five days before the illness, and the other six cases are attributed to accidents. The following is the nature of the accidents:—(1) Stunned by a fall two days before. (2) Fainted at school after being beaten; vomiting shortly after; attacked with pneumonia the following day. (3) Struck on back of head with a brush. (4) Child fell against a wall and hurt her side; this side was the one afterwards attacked with pneumonia. (5) Five days previously the head had been cut. (6) Attributed to an accident.

That chill may be a cause of pneumonia—so far as we can define a cause—no one will probably doubt. A few years ago a striking example of this came under my notice at the St. George's and St. James's Dispensary. A girl, aged 15, who was in good health, went to a school treat. After running about a great deal, feeling warm, she sat down, and then felt a *chill*. During the night she had a rigor followed by vomiting and great pain in the right shoulder. When I saw her, twenty-four hours later, her temperature was 103°, she was expectorating rusty sputum, and had every evidence of right apical pneumonia.

Before leaving this part of my subject, I would wish to in-

investigate numerically cases of other diseases in children in order to ascertain their *associations* with croupous pneumonia. Thus in 849 cases of scarlet fever I find five cases recorded; in 107 of typhoid, six: in thirty-nine of acute or subacute rheumatism, one; in 100 cases of heart disease, three; in twenty-nine of nephritis, two; and in thirty-four of diphtheria, no case was recorded.

We now consider the question of *age*. At what age in children is pneumonia most frequent? What is the earliest age when it may occur? In order to answer this question a numerical investigation of 220 cases has been made.

A brief survey of Chart I will show that, while croupous pneumonia may occur at any age from one to fifteen, so far as this enquiry shows, it is more frequent at the ages of five and nine; and it may be interesting here to mention that in a somewhat similar investigation to this (but made merely for the personal acquisition of knowledge) of 200 cases of typhoid fever in children, the highest number of cases occurred during the fifth and ninth years of life. A further survey will show that fifty cases occurred under the age of five years, 120 between five and ten, and fifty between ten and fourteen.

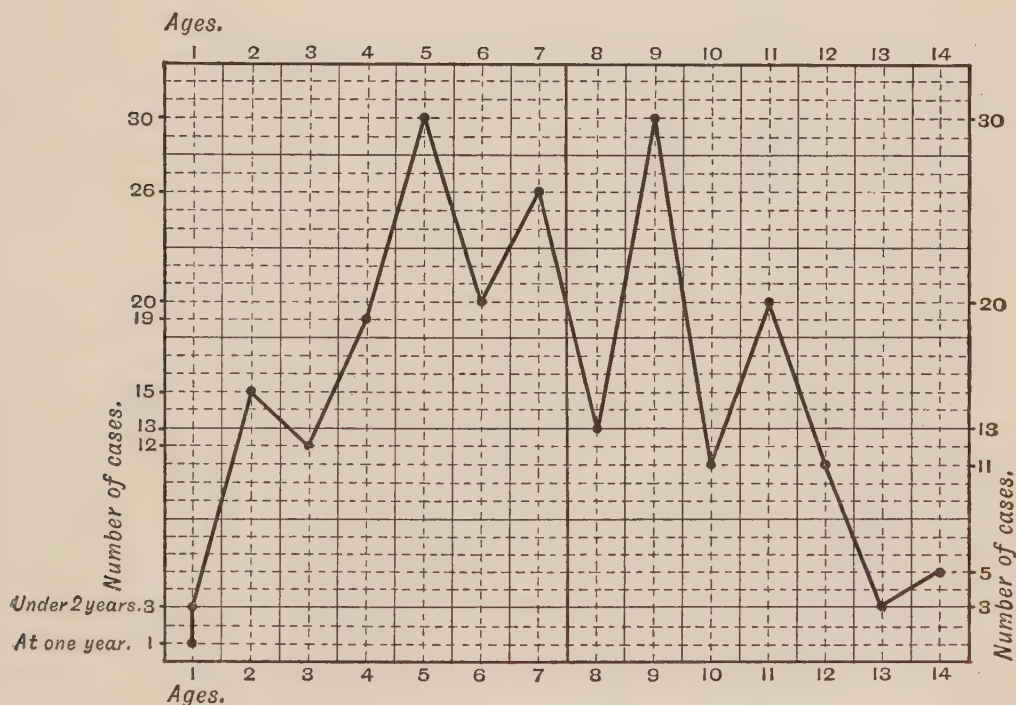


CHART I.—Chart showing number of cases at different ages in 220 cases.

In Chart II, where the cases are arranged in periods of five years, we find that there is a noticeable sudden decline instead of

a rise as age advances, and it occurred to me that this might be due to the fact that after the age of ten many children cease to be children, and being regarded as adults, go into the general hospitals. To test this I made a diagram (Chart II) from the 708 cases already referred to, and find that instead of lessening in num-

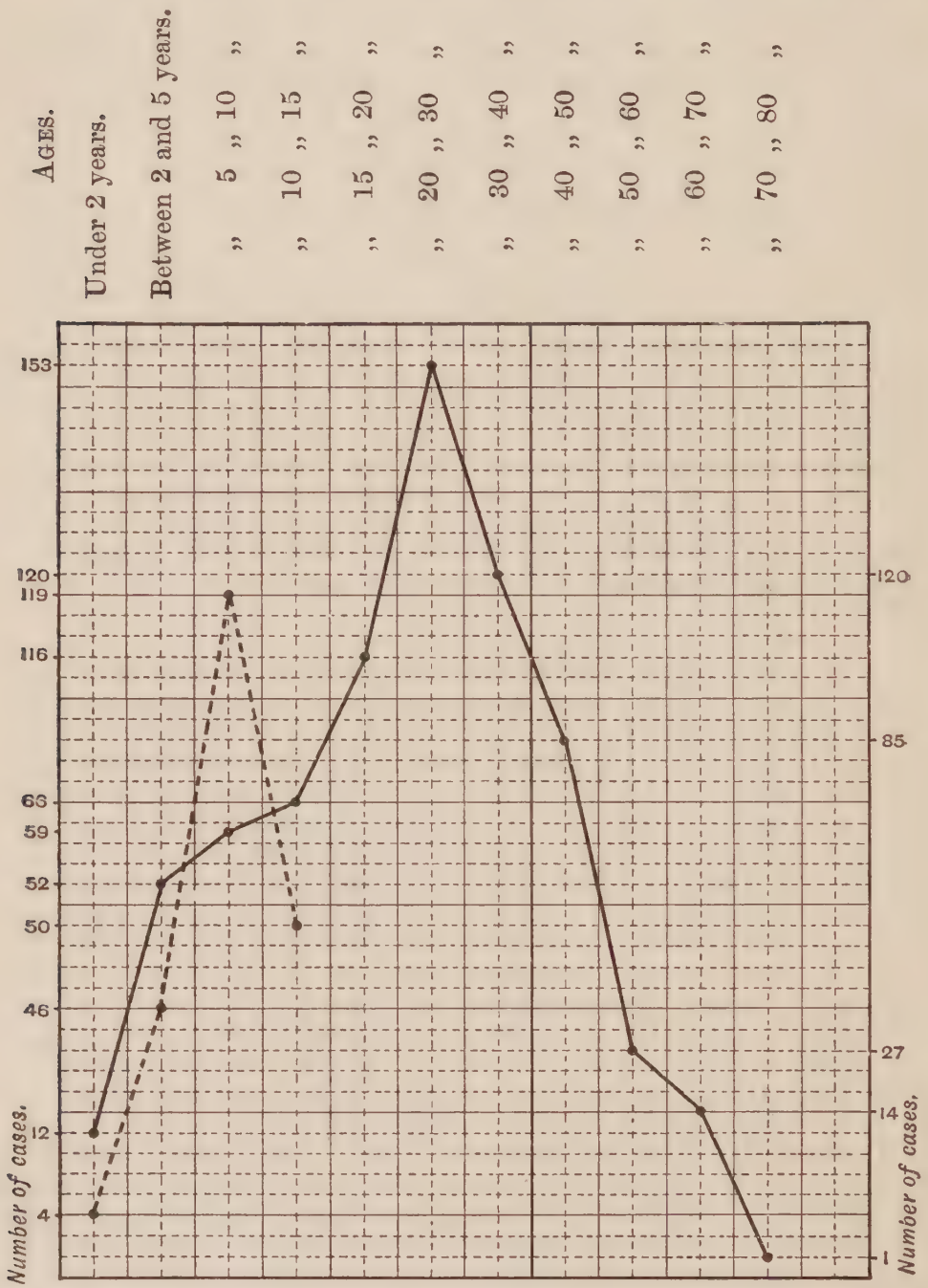


CHART II.—Dotted lines show age curve in 220 cases of children, and show apparent fall between 10 and 15. The continuous lines show age curve in 708 cases of children and adults, and show rise to be continuous till between 20 and 30, then reaching maximum and afterwards falling.



bers the proportion of cases gradually rises till between the ages of twenty and thirty; this is of some importance, because some writers state that after the age of five the frequency lessens, to rise later on at about the age of twenty: it may be so, but these figures scarcely justify the statement. Before leaving the question of age it should be mentioned that, while one year is the earliest age considered in this investigation, Barthez and Sanné state that croupous pneumonia occurs in children six weeks, four months, or six months old, and that such is seen in hospitals where children are admitted under two years of age. The youngest case that Hensch\* mentions in his analysis is one and a half years; he thinks it is common between the third and twelfth years.

*Sex.*—In 171 cases the males numbered 101, and the females seventy. Barthez and Sanné record occurrences in boys more than in girls.

We now come to the *early manifestations* of the disease. Is it sudden or gradual? To ascertain this fifty-six cases have been examined, and I find that of these the onset was sudden in thirty, gradual in ten, and not mentioned in eight. Of the ten cases showing gradual development, the period of time varied. In one case the patient was described as ailing for three weeks—in this instance there was chronic valvular disease of the heart; in another case of Pott's disease the patient was ailing for one month, but in other cases, with no attendant disease, only from three to six days.

#### SYMPTOMS.

The next point for consideration is the symptoms of invasion. *What physiological functions are disturbed by its inroad, or what are the indicators of approaching pathological change?*

To ascertain this, the records of 220 cases have been examined, and I will first of all refer to these varied symptoms in order of frequency, and then append figures representing the number of cases in which each occurred. They are as follow:—

\* 'Diseases of Children,' New Sydenham Society's translation.

Vomiting .....	134	Expectoration .....	5
Cough .....	117	Convulsions .....	3
Pain .....	111	Sweatings .....	3
Anorexia .....	75	Sore throat .....	3
Headache .....	68	Fits .....	2
Thirst .....	52	Wanderings .....	2
Dyspnœa .....	50	Coryza .....	2
Cold shivers .....	34	Epistaxis .....	2
Feverishness .....	20	Hæmoptysis .....	2
Drowsiness .....	14	Startings .....	1
Delirium .....	11	Rash all over .....	1
Rambling .....	9	Rash on face .....	1
Diarrhœa .....	7	Passage of <i>Lumbricoides</i> ...	1
Rigors .....	5	Constipation .....	1
Restlessness .....	5		

From the list we learn, at least so far as this investigation is concerned, that vomiting, cough, and pain are the most frequent symptoms threatening croupous pneumonia. For a moment I will compare this with the analysis already referred to, made by Dr. Hadden and others, when 518 were adults and 189 children, and there the most frequent symptoms were pain, rigors, vomiting, cough; thus, if we regard these as symptoms in adults, we find that vomiting,\* cough, and pain are common symptoms both in adults and children, although numerically their relative frequency is different.

We further learn that convulsions and fits are not common symptoms of invasion—and this agrees with the opinions of most writers on children's diseases. The late Dr. Wilson Fox, however, in his article on acute pneumonia, speaks of convulsions as being common in children. We further notice that rigors may occasionally occur in cases arising among children. The ages of the five patients in whom such occurred were four, eight, eleven, and twelve respectively, and the ages of the patients when expectoration and hæmoptysis were present are, as to expectoration, two at five, and one at seven, ten, and twelve; and as to hæmoptysis, four and fourteen; in neither of these cases was there any phthisis or tuberculosis mentioned as being present. Rigor, convulsions, and fits might almost be regarded as accidents in the outset. Drowsiness was mentioned, as we see, only on fourteen occasions,

\* The late Dr. Todd, in his 'Lectures,' p. 273, says: "Vomiting is a rare symptom in pneumonia." This he deduced from observations on about 90 cases.

and I draw special attention to this, as Dr. Sturges\* found it a constant symptom in twenty cases observed by him, and says, "Drowsiness is a very characteristic symptom in the pneumonia of children."

To enumerate symptoms merely in order of frequency is but a poor exercise of the mind in the study of symptomatology, so I will ask your attention to some further observations. If we group these individual symptoms into those representing disturbance in the various systems,† we find that in the *alimentary system* we have vomiting, thirst, anorexia, sore throat, pain (being a common attribute to all systems, I repeat it in each), diarrhoea, constipation; *respiratory system*, cough, dyspnoea, expectoration, hæmoptysis, pain; *nervous system*, shivers, rigors, startings, convulsions, fits, delirium, rambling, wandering, restlessness, drowsiness, headache, pain; *cutaneous system*, sweating, rashes; *nose*, epistaxis.

We thus learn that all the more important systems, with the exception of the circulatory and urinary, may be disturbed at the outset of croupous pneumonia, and, moreover, we notice that in point of variety the nervous system is the one mostly disturbed. But it will be necessary for us now to study the symptoms in regard to their isolation, association, and combination, and not in relation to systems. In so doing I find that vomiting may occur as the only symptom of invasion, or it may occur in association with one of the other symptoms, or in combination with one, two, three, or more of the other symptoms named; of cough, headache, and pain, the same may be said. Dyspnoea was not noted in isolation, or as the only symptom, but was in all cases either in association or combination with other symptoms; and thus we learn that the one symptom above all indicative of lung disease, but which is not present as a symptom of onset even in half the cases, may, when it is present, be associated with symptoms of both nervous and alimentary disturbance.

Here I must pause a while and say somewhat more in reference to *pain*, for as a symptom it is frequently referred to the part affected, and when so is valuable in diagnosis. The question arises, *Where* is pain usually seated when complained of at the

\* 'Pneumonia,' Sturges and Coupland.

† According to the 'Nomenclature of Diseases,' Royal College of Physicians of London.



onset of croupous pneumonia, and what is the relation between the situation of the pain and the part of the lung affected? I will consider the two together. In the fifty-four cases the pain was referred to the *sides* only, and in these instances

The left base	was affected in	24 cases.
The left apex	„	4 „
The right apex	„	8 „
The right base	„	14 „
The right middle lobe	„	1 „

In one case the pain was referred to the right side when both bases were affected. In another case to the left side when both bases were affected. In one case pain was referred to the left side, and the right apex was affected. In one case to the right side and abdomen, and the right apex was affected, and in another case to the right side and head, when the right base was the seat of the pneumonia.

In the six cases in which pain was in the chest

The right apex	was affected	twice.
The right base	„	once.
The left apex	„	once.
The left base	„	twice.

In one case, when pain was referred to the chest and head, the left apex was the seat of the disease, in another of pain in chest and throat the right base was affected, and in a third case of pain in chest and limbs the left base. Pain was spoken of as being *over the heart* in two cases, when the left base was the seat of pneumonia in one case, and the left apex in the other.

Pains in the head (not headache) occurred in two cases, and the right apex and right base were affected respectively. Pain in the head and limbs in one case and the disease was in the left apex. Pain in head and back in two cases, and the disease attacked the right apex and right base respectively. Pain in head, back, and sides once when both bases were affected. Pain in head, neck, and side, with the left base affected. Pain was described as being *all over* in one case when the left base was affected; and pain in the limbs once, and the left base was then the seat of the disease.

Pain was described as being in the abdomen in fourteen cases,

and when so the parts of lung affected were as follows:—Right apex in two cases; right base in six cases; and left base in six cases. Pain was referred to the epigastric region once when the left base was affected.

From these facts we learn that in most instances when pain occurs as a symptom it manifests itself in the sides, and that when so it is seated on that side where the lung is afterwards found to be affected. We also find that pain may be in the chest, directly over the heart, in the head, limbs, or abdomen—pain over the heart indicating left-sided pneumonia, in the abdomen mostly the bases; and when pain is in other parts various parts of the lungs may be the seat of disease.

#### DIAGNOSIS.

First, are we able to diagnose croupous pneumonia from our knowledge of the symptoms attending its early invasion? Are these symptoms either alone or in association or in combination peculiar to this disease? Here we call into exercise another faculty—comparison. And, for the sake of comparison, I have read over the notes of a number of cases of scarlet fever, typhoid, meningitis, and acute nephritis. I also call to mind the fact that vomiting may be associated with intestinal lesions, or be a symptom of restoration of function, and that cough also may be but a reflex result of ordinary constipation. Now, vomiting, as we have seen, may occur as the only symptom of invasion, and as such be sudden in its onset; but it may also occur suddenly as a symptom of restoration removing some irritant from the stomach, or it may be due to acute intestinal obstruction; and it may indeed indicate the onset of scarlet fever, typhoid, or meningitis. Vomiting also, when associated with diarrhoea, is not an unknown symptom at the invasion of typhoid fever or acute nephritis, neither is it infrequently met with in association with either sore throat, rigor, or shivers, in incipient scarlet fever. Headache or shivers, separately or together, with or without diarrhoea, are frequent symptoms of typhoid, while rigors with a sudden onset are not unknown as preceding it. Shivers may also usher in acute nephritis, and vomiting with headache and drowsiness precede the onset of meningitis. The symptoms most common to croupous pneumonia, then, appear to be cough, pain, and dyspnoea, and the



latter is the most important; for the cough we can hear; the pain is reported to us by the sufferer; but the fact that dyspnoea is present is oftentimes only revealed to us by observation, and amidst the numerous early symptoms this may be frequently overlooked. It is thus rather to the association and combination of symptoms that we must look for guidance, and it is better to be wrong from reasoning than correct by guessing. But even after reasoning to the best of our ability we shall frequently be obliged to defer a definite and decided diagnosis until, the disease being firmly established in its site, we can find sure and certain signs of its existence—these I will presume are physical, and the question then arises, how soon may we expect to find such signs? For the present, however, I will defer the answer, and consider the *temperature* in croupous pneumonia.

On reading over the notes of cases yielding material for this investigation, it is obvious that croupous pneumonia is a disease in which (in the largest number of cases) the temperature is high with slight morning remission and evening exacerbation until it falls either suddenly (crisis) or gradually (lysis); and such being the case, the question naturally arises, what is the usual temperature in pneumonia, how high may it rise without creating much alarm, and how long will it remain high? First, then, as to height of temperature before the crisis. In 152 cases I find that the average maximum daily temperature varies from  $100^{\circ}$  to  $105.4^{\circ}$ . By dividing into tens the points gained, in fifty-eight cases it varies from  $103^{\circ}$  to  $104^{\circ}$ , in thirty-six cases from  $104^{\circ}$  to  $105^{\circ}$ , and in thirty cases from  $102^{\circ}$  to  $103^{\circ}$ . In a few cases it ranged from  $105^{\circ}$  to  $105.4^{\circ}$ , and the least frequent variation is from  $100^{\circ}$  to  $101^{\circ}$ . It must be remembered that in some cases the temperature does not rise above  $100^{\circ}$ , when at the crisis it usually becomes subnormal for a time. This leads me to a consideration of the temperature at *crisis*. What is the highest temperature on the day of crisis? In 130 cases examined the temperature varied from  $100.2^{\circ}$  to  $105.4^{\circ}$ , being in thirty-nine instances  $103^{\circ}$  to  $104^{\circ}$ , in twenty-nine  $102^{\circ}$  to  $103^{\circ}$ , in twenty-seven  $104^{\circ}$  to  $105^{\circ}$ , in twenty  $101^{\circ}$  to  $102^{\circ}$ , in eight  $105^{\circ}$  to  $105.4^{\circ}$ , and in seven  $100.2^{\circ}$  to  $101^{\circ}$ . Thus we see there is practically no difference between the average maximum daily temperature and that of crisis, while there is a difference in individual cases. While usually we have the temperature persistently high till the crisis, we may have a pseudo-



crisis—the temperature falling to normal, then rising to say  $104\cdot2^{\circ}$ , to fall to normal a day later than that of the true crisis. After the crisis the temperature may be high, and continue so for some time; this happened in one instance, when the left ear commenced to discharge and the temperature fell to normal. There is another point in reference to the temperature. Is the temperature range higher in cases when the apex only is affected or when the base only is involved? So far as this investigation shows there is no great difference, but if anything the temperature from basic pneumonia is the higher, as is shown by the following:—

In twenty cases of apical pneumonia the average daily temperature was—

In one case it varied from $101^{\circ}$ to $101\cdot8^{\circ}$			
„ six cases	„	102	„ $102\cdot4$
„ nine	„	103	„ $103\cdot8$
„ one	„	104	„ $104\cdot5$
„ three	„	105	„ $105\cdot2$

In twenty cases of basic pneumonia the average daily temperature was—

Two cases varied from $101^{\circ}$ to $101\cdot8^{\circ}$			
Three	„	102	„ $102\cdot8$
Eight	„	103	„ $104\cdot3$
Five	„	104	„ $104\cdot3$
Two	„	105	

Yet another point in reference to temperature is of interest, namely, the relation of *delirium* to temperature.\* Is the temperature higher in cases when delirium is present? So far as this investigation shows the difference is only in individual cases, when the temperature is a degree or so higher in cases of delirium.†

In twenty cases of delirium the average daily temperature was as follows:—

100°	in one case.
101·6	„
102·7	„
103	„
103·1	„

\* See “Some Statistics of Pneumonia,” by A. Money, ‘Trans. Roy. Med. Chir. Soc.,’ vol. lxi.

† Although in this paper I do not enter into causes, it may be mentioned that

103·3° in two cases.

103·5 in one case.

103·6 in two cases.

103·7 „

103·8 „

103·9 in one case.

104·4 „

104·5 in two cases.

104·8 „

In twenty cases having no delirium the average daily temperature was—

100·1° in one case.

101·2 „

101·3 „

101·6 „

102 „

102·3 „

102·4 in three cases.

103·5 in two cases.

103·6 „

103·7 „

104 in one case.

104·1 in two cases.

104·2 in one case.

104·6 in two cases.

It is of interest also to observe that in cases of double pneumonia the daily temperature is not in excess of the highest average daily temperature when only one lung or part of a lung is affected. Now is a high temperature with slight morning remission and evening exacerbation the only type of temperature met with in croupous pneumonia? And if we meet with any other type, is it due to some other direct cause or merely to the accidents of the disease?

Hectic temperature is frequently met with, it being, I find, recorded in thirty cases out of 220. It may be pre-crisial or post-crisial. The temperature may be of a hectic character throughout,

I regard delirium in pneumonia as due to toxic causes not necessarily causing any additional rise in temperature or increasing the danger of the case.

becoming normal after the crisis. It may be hectic at the commencement, and subsequently follow the type of normal pneumonic temperature to fall to normal at the crisis, or it may be at first of ordinary pneumonic type and then become hectic. This may be merely accidental, but in two cases it was associated with accumulation of purulent fluid in the pleura. But while we have a pre-crisial hectic, it is more often met with as post-crisial, this being so in twenty of the thirty cases. Hectic temperature usually commences a day or two after the crisis. It may be present for several days and then change to normal, or it may be due to post-crisial pleurisy or catarrhal pneumonia. In one case the temperature became hectic after the crisis, and was so till the forty-eighth day, when 8 ounces of pus having been removed it became normal.

The temperature may be of a remittent character (not hectic); this was the case in two instances, one after crisis and one after lysis. In one case before lysis (which was on the seventh day) the temperature was subnormal on the fourth day, then varied from  $99^{\circ}$  to  $102.3^{\circ}$  till the seventh day, when it fell with great rapidity until the tenth day. The temperature may be intermittent, but this is very unusual. In a case complicated with Potts' disease the temperature remained hectic till the fiftieth day.

Having considered temperatures, I next proceed to ask, *when may crisis occur?* Of 176 cases I find that crisis occurred in the majority of instances between the fourth and the tenth day, but in most cases on the sixth and seventh days. It may occur any day from the third\* to the eighteenth.

Chart III shows the number of cases on each day. I will now ask, Is there any difference as regards the days of crisis in *basal* and in *apical* pneumonia? In twenty cases of *basal* pneumonia I find that the crisis occurred between the fourth and eighth days in fourteen cases, between the eighth and eleventh in four cases, and on the eleventh and thirteenth days in one case. And in a similar number of cases of *apical* pneumonia crisis occurred between the fourth and eighth days in ten cases, and between the eighth and eleventh days in ten cases. Thus in a given number of cases the crisis in *basal* pneumonia occurs on or before the eighth day in more

\* It would appear that crisis may take place as early as the second day. The notes of the cases from which I deduce this fact are not quite reliable, so the cases are not put in the investigation.



cases than in a similar given number of cases of apical pneumonia; yet in basic pneumonia the crisis may be longer delayed.

In the twenty-two cases ending by lysis it lasted from two to seven days, being in most cases three and five days. Now I will revert to the question, When, that is, how soon after the symptoms of invasion, may we expect to find physical or positive signs of croupous pneumonia? To begin with the well-known and readily recognisable signs—dulness, increased vocal fremitus, tubular breathing, and increase of vocal resonance (only let it be observed that in children increase of vocal fremitus and vocal

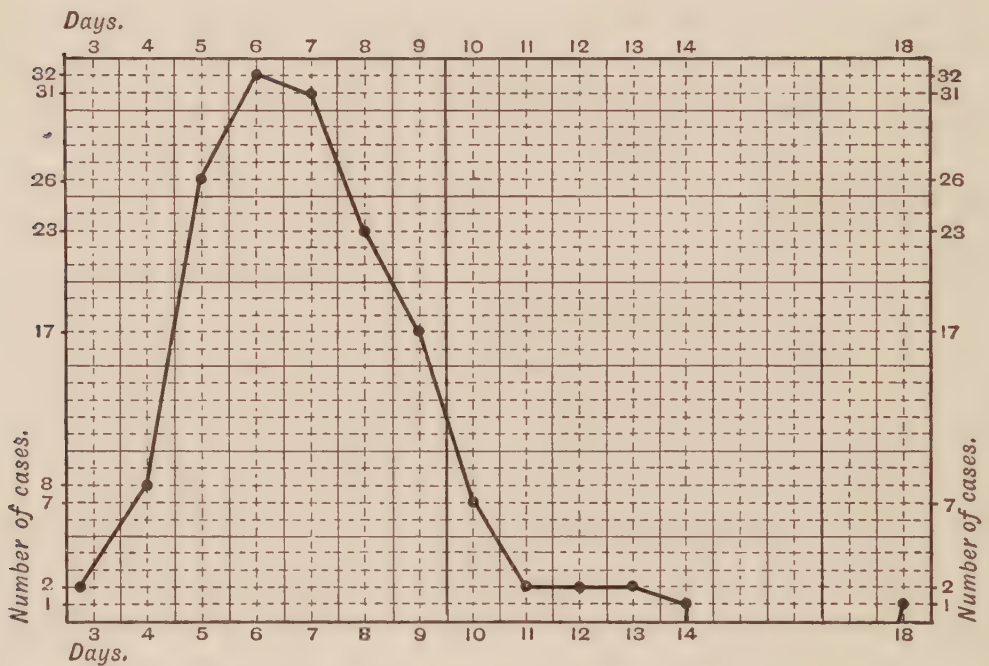


Chart III.—Showing days of crisis in 154 cases.

resonance, although constantly present, are not such constant factors as in the adults)—we find such may be present within thirty-six hours after the symptoms of invasion; but in many cases they are not present till the fourth day. It is of some interest to note that such symptoms as vomiting, cough, and pain are regarded as of little importance; indeed to the many they are everyday symptoms, and thus the lungs are often not examined till late, so that much information on this point is still wanting. It is, however, a fact that in some cases the physical signs above named are absent not only till the fourth day, but also till the fifth, sixth, or even seventh, being *recognised* in some cases only a day or two before the crisis. I would, however, ask, Are such signs, so

plainly evident to the student just commencing the study of physical diagnosis, necessary for the purpose of diagnosis? Are they not rather corroborative so far as diagnosis is concerned, and should they not be regarded as signs for *prognosis*, to show us how much of the lung is diseased, and the stage of the disease, and so help us in our estimate of the probabilities of recovery in each individual case? If we regard them as such, what then are the *physical signs* of pneumonia? One of the earliest signs is a dull tympanitic note on percussion (this is due to a relaxation of the lung tissue) which is often present before any marked variation in the breath-sounds is discernible. Then we may have deficient resonance with *weak* breath-sounds, or a deficient resonance with harsh or distant tubular breathing, and we may have, when the disease is fully advanced, dulness, &c., with weak breath-sounds, and, under exceptional circumstances, the breath-sounds may be vesicular, or they may be so indistinct as to be described by some as absent.

But in this paper I must forego further remarks on a subject which interests me greatly, and I will ask, *what part of the lung, and which lung*, is most frequently the site of croupous pneumonia? In 146 cases I find that the left base was the site of the pneumonia in forty-six cases, and the right apex in thirty-five, the right base in twenty-three, and the left apex in ten. Both bases were affected in six cases; both apices in one case. The right lung in six cases, and the left also in six. The right middle lobe in four cases, the left axillary region in one case. The right base and left apex in one case. The right apex and left base in five cases. The left lung and right middle lobe in one case, and the right lung and left base in one case. From these facts it will be seen that the bases are more often affected than the apices—the bases being affected in sixty-nine instances, and the apices in forty-five. It is, moreover, noticeable that the left base is the most frequently attacked, and the right apex next, and that the right lung is slightly more frequently attacked than the left.

Having then considered the physical signs and regions of lung affected, I will ask your attention to *the duration of the existence or presence of physical signs after the crisis*. We have seen that the temperature falls after the crisis, and we may therefore ask, What effect has the crisis on the physical signs? In reading over the notes of 157 cases, one may say that crisis has no marked effect

upon the physical signs; they still exist as they did before; and further observation of these cases shows us that physical or abnormal physical signs, one or more of which are met with in pneumonia, may exist for a long time after the crisis, and that the earliest day—and then in only one case—that such ceased to exist was the seventh. Thus in 157 cases physical signs were recognised in

110 cases from the	7th to the 20th day after crisis.
35	„ 20th „ 30th „
10	„ 30th „ 40th „
2 cases till the	44th and 48th days respectively.

Hence, if we notice abnormal physical signs persisting after the crisis for some days, we need not feel unnecessarily anxious, or try to divine some special cause. Expansion of the *alæ nasi* in respiration being the first objective sign, I will at once notice the *modifications of respiration* in croupous pneumonia. In twenty-two instances when this information was recorded, the respiration was found to be much increased in frequency, the highest number per minute before the crisis being 68, the lowest 32. At the crisis the rate of respiration falls like the temperature, in one instance being 64 at the crisis, but on the day following 26.

In quality the respirations are short, and at the crisis there may be great distress with breathing, either alone or in association with cyanosis, neither of which, however, necessarily indicates fatal termination, while croupous pneumonia may run its course unattended by other respiratory signs or symptoms than those already spoken of; it does not always do so—we may have expectoration. This was present among the cases under consideration eight times, the respective ages being one at six, seven, and nine, two at eleven, and one at ten, twelve, and thirteen, being *rusty* in seven cases, and *mucoid*, tinged with blood, in one case.

*Cough*, which occurred 117 times as a symptom of invasion, was recorded in only one instance during the attack, and then as “violent at the crisis.”

*Pleurisy* existed in sixteen cases; the earliest date given as to its being observed was the fourth day; in some cases it was not noted till the eighth. Effusion occurred in one instance, and it was purulent in three, pus being removed from the chest on the tenth, thirteenth, and forty-eighth days respectively. In thirty cases of



empyema, the histories of which I have examined, I find only two cases recorded in which it was referred to as following croupous pneumonia.

Bronchitis and catarrhal pneumonia also occurred as complications. Gangrene of the lung was observed in one case out of 220. The patient, a boy aged twelve, died on the sixth day; there had been high temperature ( $104^{\circ}$ ), slight expectoration, and signs only of pneumonia.

*Epistaxis* occurred during the course of the pneumonia in one case.

As in its invasion so during its course other symptoms may be disturbed—one might say all the symptoms. Hence in the *circulatory system* we find the pulse increased in frequency, varying from 80 to 160; like the respirations, it increases in frequency as crisis approaches, and then lessens—in one instance being on the day of crisis 148, the day following 76. The pulse may also be irregular. Apart from changes in the pulse there may be signs indicating changes within the pericardium or the heart; hence friction at the base was observed in one instance in 220 cases. The patient was suffering from aortic and mitral disease, due to a former rheumatic attack, when friction (pericardial) was heard on the second day after the patient had been under observation.

Endocardial murmurs may also arise during the pneumonic attack, such being noted in three cases of the 220: in one a murmur at the base with reduplication of the second sound was heard on the fifteenth day, in another at the base on the nineteenth day, and in a third at the apex on the sixteenth. The two murmurs at the base cleared up, but the one at the apex was present on the day of discharge. In four cases endocardial murmurs were heard at the apex on the day of admission; in one instance a note is made to the effect that the murmur disappeared on the thirteenth day.

*Alimentary System.*—*Vomiting*, it will be remembered, as the most frequent symptom of invasion, occurs also during the course of the disease, but with much less frequency, being noted on only eight occasions in the whole 220 cases. It may continue from invasion to crisis, or it may be pre-crisial only, or pre-crisial and crisial, or crisial only, when it may be very violent. It may be absent at the onset of the attack or occur only subsequently.

*Diarrhœa* occurs almost as frequently during the course of the

disease as at its commencement. At the onset it was recorded in seven instances; during the course I find it recorded as having occurred six times. Like vomiting it may be pre-crisial only, or pre-crisial and crisial, or crisial only, or it may be post-crisial, and it may be associated at the crisis with vomiting.

*Tonsillitis* occurred after the crisis in two cases, on the eleventh and nineteenth days respectively.

*Aphthæ* were noted in one case, and this case recovered.

*Urinary System.*—I here refer only to the presence of albumen, which was recorded as being present in fourteen cases. Before, however, speaking of albumen, it should be mentioned that in one case there was nephritis with anasarca on admission; this condition lasted till the day following the crisis, when it is reported as “all disappeared.” Albumen was present on admission in seven instances; it is noted as disappearing in three cases; in one, however, it was present till the twenty-third day. Albumen was found in the urine *after* admission—not being present on admission—in seven cases; in six it was noted between the third and fifth days, but in one not till after the crisis.

*Integumentary System* (under this heading I include conditions of the skin).—Herpes was noted in thirty-seven cases out of 220. This developed mostly on the lips and at the angle of the mouth, but other seats of attack were as follows: tip of nose and upper lip, alæ nasi and cheek, left cheek and angle of the mouth, left cheek only, left arm, wrist, and angle of the mouth. The rash of measles was noted in one case on the day of crisis—the seventh. A diffuse erythematous rash, thought at first to be scarlet fever, was noted in one case, and erythema nodosum on the eleventh day in another case. The skin was cold and clammy at the crisis in several cases, and sweating took place at the crisis in six cases. Sweating also occurred during the attack before the crisis in a few cases, and after the crisis in one. Œdema was noted in two cases.

*Nervous System.*—I will here first refer to delirium. As a symptom of invasion we noted it in eleven cases, but as a symptom during the course of the disease it is of much more frequent occurrence; hence I find it mentioned in no fewer than thirty-four cases. Like other symptoms already referred to, it may be pre-crisial and crisial, but it was in no instance recorded as being post-crisial. In twenty-cases it was pre-crisial, and in one case, when it occurred on the second day after admission, the

patient was very violent. It was both pre-crisial and crisial, in one case commencing two days before the crisis. In one case it was a symptom of invasion, and continued till the day of crisis. In two instances in which it was not present at invasion it set in shortly after, and continued till crisis. In four cases it set in with lysis, and lasted for two days.

I must ask your attention to a very interesting inquiry—that is, as to the part of the lung attacked when the disease is accompanied by delirium, and I shall only refer to the relative number of cases in which the disease was apical or basic. I find that of twenty-eight cases where these facts are recorded, in sixteen the apices were affected, the right apex being so in fifteen of the sixteen cases, and that the bases were affected in twelve instances—in seven the left base, and in five the right; and I may here mention that in the ten cases where delirium was mentioned as being present at the onset, the apices were affected in four, and the bases in six cases. Only one of the cases in which delirium was present died.

Of other nervous phenomena in the 220 cases, I find retention of urine for twenty-four hours just before the crisis, involuntary passage of the fæces till the ninth day, which was the day of crisis, and spasm of the posterior cervical muscles occurring on the day of crisis.\*

I must mention that the pneumonic attack may be of a relapsing or intermittent type;† one such case was recorded amongst the 220.

Having now gone over the onset, diagnosis, and course of this extremely interesting disease, the next step would naturally be to investigate those symptoms indicating recovery or the reverse; but time does not admit of my bringing before you what may be termed the elements of *prognosis*. The fact, however, may be mentioned that of 242 cases only nine died, thus showing that croupous pneumonia in children is not a disease which is frequently fatal, and of the thirty-two cases when delirium was present, only one died.

\* Hemiplegia may occur during the pneumonic attack, but no such case was observed in the 220 (*vide* “Hemiplegia in Typhoid,” ‘Trans. Clin. Soc.’ vol. xxvi).

† I have not myself met with this type in children, but have seen one case in an adult. A case is graphically described by Sir Andrew Clark in the ‘Transactions of the Medical Society,’ 1884.



Permit me to emphasise the absolute necessity of not only knowing how to diagnose, but also of knowing all the various forms and types of disease—in fact its natural history. Without this knowledge, which of necessity presupposes the capacity for diagnosis, how can we make a prognosis? And unless we can forecast how can we treat disease rationally, for how are we to know that that which we treat requires treatment? I would much like to have deduced facts from the treatment of the 220 cases, especially in reference to the use of poultices or ice. I have heard it said, “Children bear the application of ice well, therefore we will try it”; but surely that fact alone should not be used to determine in its favour. Rather should we note the condition of the heart and the site of the pneumonia; for all agree—although it is not so usual in children as in adults—that death frequently occurs from cardiac failure; and this fact, it seems to me, should aid us in determining when ice or poultices should be used. Both are cardiac stimuli, but they act in different ways. One—poultices (or heat)—physiology teaches us increases the heart’s frequency, while the other—ice—slows it; and I believe an undue slowing of the heart before the crisis does harm in pneumonia, and increases the risk to life. My own view, so far as it is formed, is that ice in a left-sided pneumonia, unless used with great caution, may so slow the heart as to become a cause of death (so far as my own judgment serves me I have seen this occur). There are other questions, also, notably the question of stimulants, to determine in what cases, or under what conditions, they are absolutely essential for the recovery of the patient, and also when the undue administering of them helps to produce a fatal result; when and how the temperature should be interfered with by treatment, and also when a case, and what class of cases, would be better for being left alone without any treatment beyond some local application, a warm bed, and suitable diet, but none of these points can be touched upon on the present occasion. They can, however, only be decided by our first knowing all about the disease, its normal and its abnormal types, and the effect of differing constitutions upon its course. Thus if I have—as I fear may be the case—gone too much into detail, my excuse is the necessity of ascertaining facts for the guidance of the judgment. To do this demands detail, and towards it all our studies should tend.

Dr. DE HAVILLAND HALL wished to know whether Dr. Hawkins was prepared with any statistics as to the frequency of catarrhal pneumonia in children as compared with croupous. He quoted an American writer, to the effect that in children under five years of age pneumonia was almost always catarrhal; between five and fifteen the incidence was about equal, and above fifteen croupous pneumonia was the rule.

Dr. ALEXANDER MORISON remarked that he had observed as a clinical fact, with indisputable distinctness, the sudden origin of croupous pneumonia by metastasis of coryza in a child under four years of age. He believed that it was possible that other cases might originate similarly, and that, medical assistance being sought when the graver malady originated, no trace of the preliminary affection might be found. He agreed with the authority mentioned by Dr. de Havilland Hall, that croupous pneumonia under five years of age was a comparatively rare affection, that is, very considerably rarer than catarrhal pneumonia. Crisis in children he had observed to be better marked than in adults, and usually to occur about the fifth day. He did not place much confidence in a mere statistical estimation of clinical facts, unless some well-established physical factors underlay the conclusion. He made this remark in reference to Dr. Hawkins's statement that there was a considerable preponderance of right-sided pneumonia. While in a case of acute or primary affection, he did not think that the site of inflammation was important; he believed that there were *a priori* reasons for believing that a pneumonia arising in the course of a debilitating disease such as typhoid fever would select the right rather than the left lung base. Referring to Dr. Hawkins's remarks upon the use of ice in pneumonia, he believed that the heart could be regulated as well by the application of ice to the forehead as by its application to the chest or precordia, and that its use, which he did not question, was safer when so employed. While he was not averse to the guarded use of alcohol, he believed its danger lay in blunting the motor reflexes, especially the cardiac reflex, both directly by ordinary excessive action, and indirectly by its anæsthetic action on the cardiac centre. Speaking of the employment of digitalis in pneumonia, he regarded its use with great suspicion in the pneumonia of adults, but believed it was at times of use as a cardiac stimulant in children, and less dangerous in them because of their more elastic vascular system. He had been converted to this opinion by Dr. Cheadle.

Dr. P. MURRAY BRAIDWOOD remarked that croupous pneumonia was to be distinguished from catarrhal pneumonia rather by its *post-mortem* appearances than by clinical symptoms. In his experience, in many instances, especially in children, whose chest walls are thinly covered while their pulmonary apparatus is proportionately very expansive, it was impossible at the onset of the disease to diagnose croupous pneumonia. Hence it is that the onset of this disease is said to be gradual. In fact this pathological condition is very commonly not recognised till resolution has fairly set in. He agreed with Dr. Hawkins that rigors were very seldom initiatory symptoms of this disease. Pain was often present, but was no aid to diagnosis. The nervous system he regarded as always seriously deranged by croupous pneumonia. Cough was necessarily always present. He agreed with Dr. Hawkins that the clinical thermometer does not aid us much in diagnosis. As regards treatment, his experience lead him to recommend strongly counter-irritation by the constant use of hot poultices to the back of the chest (ice was dangerous, especially to children) and a hot bath daily to encourage diaphoresis. The employ-



ment of alcohol, he considered, should be as a sedative rather than as a stimulant.

Dr. PASTEUR alluded to the great difficulty there was in distinguishing *clinically* between some cases of broncho-pneumonia and cases of croupous pneumonia, and hoped the author of the paper would mention the criteria on which he had mainly relied in drawing a distinction between them.

Dr. WALTER CARR said that there were as marked differences in type in the croupous pneumonia of children as in that of adults, and, therefore, mere averages deduced from a large number of cases were comparatively valueless, the extremes being too great. In previously healthy children, however, the disease generally ran a very definite course, with a sudden onset, a continuously high temperature, with little or no remission, and in a few days, usually less than a week, a very sudden crisis, very often in the night. Recovery, as a rule, was rapid and complete, perhaps the most frequent sequela being empyema. A hectic temperature or a gradual fall to normal was strongly suggestive of catarrhal, rather than of croupous, pneumonia. No special treatment seemed necessary, a fatal issue being rare, except in children who were also suffering from one of the acute specific fevers or from marasmus. Although not common in children under five, the disease might occur, and run a typical course even in infants of less than a year old.

Dr. HAWKINS, in reply, could not agree with those who regarded croupous pneumonia as rare in children—its rarity consisted in its not being diagnosed, and thus being confounded with a “feverish attack,” or some other disease, as “meningitis.”

---

*April 10th, 1893.*

## AN ADDRESS ON NEUROLOGY AND THERAPEUTICS.

By W. R. GOWERS, M.D. Lond., F.R.S.

MR. PRESIDENT AND GENTLEMEN,—I count it no small honour to occupy the position in which I am placed to-night, not only as the first recipient of a distinction which must ever be one most highly esteemed by the profession, but as having the duty placed upon me of inaugurating the new arrangement for the Fothergillian Prize. The duty, indeed, is one which I feel carries peculiar responsibilities, because it involves a precedent of no small importance and lays upon me the task of setting an example of no light responsibility. I wish that I could feel satisfied that the duty would be fulfilled and the example set in a manner worthy of the occasion, but I must rest content with the endeavour to do my best and with the consciousness that I may safely reckon on your indulgence.



These are, however, not the only difficulties which present themselves to my mind as encircling the duty, pleasurable though it be, which lies before me. The founder of this Society, in whose honour the prize was established, lived and worked in the days when every physician was, before all else, a practitioner of his art, and when the birth-throes of abstract medical science had scarcely begun. The practical aspect of the work of the physician never can vanish (except in the case of those pure scientists to whom the name ceases to be applicable), nor can it cease to be of pre-eminent importance, but it has changed in relative predominance, especially as regards the different periods of the physician's working life. Here, as everywhere, "the old order changeth, yielding place to new." The physician was formerly a practitioner from the very first—from the earliest days of his career—but now this is true only of some. The change has been inevitable. Science is an exacting mistress; medical science is not the least exacting, and with advancing years she has renewed her youth and multiplied her demands on the devotion of her servants. If many physicians did not devote their early years to scientific work, our knowledge could not have reached the point it has attained; the prospect of future advance could not be so bright as it is, whatever the actual degree of that brightness may be, did they not still do so.

The different character of the work of physicians in the early and middle period of their active efforts to further the progress of medical science is reflected in the character of the various societies which exist and of which they are members. The founder of the Medical Society of London, whose memory this prize will always preserve, possessed, in a conspicuous degree and throughout his career, the practical characteristics which dominated the work of the physicians of his day. The Society has always kept before it this aspect of medicine, and has thus been of peculiar service to all practitioners, a service on which it may still justly pride itself. Hence it seems to me to be incumbent on those who venture to address it, and especially on those who may occupy the post I have the great honour of occupying to-night, to keep in view this aspect of its work, confirmed by the practice of so many generations and possessing, as it certainly does possess, a high degree of peculiar utility. But with it there is associated a duty which is not, I think, always recognised or always fulfilled,

and which is yet of extreme importance. In the present day the abstract cannot be ignored. It asserts itself at every turn in scientific work. And with it another fact, of paramount importance, is asserted in tones too clear not to be heard and too distinct to be mistaken—the fact that there is no abstract work which does not at some time or in some way find a practical application. True in each department of science, it is conspicuously and almost invariably true in medicine. This application is, moreover, more frequently immediate, less frequently deferred, than in the case of most sciences. In this fact there is involved this duty, which I would ask you to permit me to emphasise. The practical application of the abstract investigations of medical science can be indicated by none so well as by those whose researches have supplied the abstract results themselves, and who can, if they be inclined to do so, at least indicate the practical applications of their investigations in a way and with an efficiency that is possible to no one who comes after them and takes up the thread of their labours in order to weave it into the texture of the practitioner's daily labour. But this requires a diversion of effort from the more attractive and perhaps entrancing continuation of the abstract work which is in hand. It is of the utmost importance that there should be no needless delay in the adoption, in practice, of every result that science obtains, and my reason for thus dwelling on what may seem somewhat irrelevant is to urge on the abstract worker the extreme importance of endeavouring, at each stage in his labours, to perceive what applications his results may have, and to point them out for the benefit of those who devote themselves to the practical application of the abstract and who might not, and probably would not, perceive them without the aid thus rendered. There are, indeed, instances in which the abstract and the practical are united in one person. Instances will occur to you without my aid, and a conspicuous example is before you in the person of your President.

I must ask you to allow me to preface my special remarks regarding the treatment of disease of the nervous system with some observations of a more general character on the pathology of those maladies, or rather on the change which has come with increased knowledge on our conceptions of their nature. In doing this we cannot separate physiology and pathology. The two subjects will not be divorced, and this is true now to a degree and extent to



which it was never true before. The more we learn of each, the more intimate, the more essential, the more radical, we perceive to be their union and their unity. Disease is health gone astray; pathology is but perverted physiology, and we can only obtain an adequate idea of morbid processes by acquiring first a knowledge of those of health. This fact is familiar enough, but it will bear repetition in connection with the minute and ultimate elements of these processes. We shall see this in a moment. Of necessity our view must be restricted to those points which are related to the topic that is before us. We cannot range at large over the whole wide subject, attractive though much of it may be and is; but the special conditions which underlie the diseases we have to treat must underlie our therapeutics, and we must consider the changes that have come over our conception of the normal processes, and which have entailed corresponding changes in our conceptions of the related processes of disease, before we can, with any prospect of success, attempt to ask ourselves even elementary questions regarding their treatment.

Without doubt the present generation has witnessed a vast change in our conceptions of the structure of the nervous system, of its functions, and of the disorder of both. From these has proceeded a necessary change in our practical thoughts,—in the measures we adopt in endeavouring to alter that which is wrong and in our efforts to substitute order for the disorder that we can observe. But a firmer and more rational foundation has also been obtained for measures that are by no means new. If we try to perceive the nature of the change in our conceptions and try to epitomise it in a brief expression, I think we should say that its essential nature has been the substitution—forgive me if I seem to use an expression of perplexing ambiguity and obscure brevity; it will clear itself in a moment—of the analytical for the synthetical method of thought, of analysis for synthesis, in our ideas alike of health and disease. Expanded into somewhat simpler terms, it may be said that the change has involved a perception of the many where before we saw the much. The function which appeared to us to be single and simple we now perceive to have had only a spurious simplicity and to consist of a complex union of distinct elements, working together or working apart, but not to be confounded, except with the certain result of being misunderstood. Here, as elsewhere, we must, as a great thinker once



said, distinguish where we cannot divide; and, I may add, we must divide where we cannot separate.

You may now observe that I have been only saying that which is familiar, but saying it in recondite phrase—a process that is apt to be annoying, but yet is not without its advantage. It tends to give the familiar a fresher aspect and to fix its features on the mind in a more effective manner. Grasp the change of conception which has taken place, in the aspect in which I have described it; realise that it has generally consisted in a substitution of the particular for the general. The gain has been immense, because the elements were seldom all united, and their partial effects produced too often insoluble difficulties when the constituents had not been discerned, and when we could only see an imperfect whole instead of perfect parts. Consider the functions of the cortex of the brain as an illustration of this truth. You can do so without my aid. I need not dwell upon its features; I need not point out the details of the illustration it affords, because these are well known to every one of you, and I have already perhaps spent too much time over that which it is superfluous to discuss. I shall have, moreover, to consider another illustration, on account of its special significance. The point to which I would bring your thoughts—that for the sake of which I have dwelt on the nature of the change which has taken place in our ideas—is the probability, or rather the certainty, that this process of analysis is far from finished. Absolutely complete we must not expect it ever to become, but we cannot even conjecture at present how far off may be the point which we may reasonably regard as that of relative completion; we cannot yet guess how far we may be from the point at which we shall reach the ultimate of our practicable analysis, the point beyond which we cannot go with the confidence that science demands and a scientific habit of thought must have.

But I must ask you to retrace our steps, in order to gain a clear conception of the nature of the processes in the nervous system in their relation to treatment. Looking back, we see that the first step in the analysis of the structure of the lower parts of the nerve centres was that the white columns of the cord were resolved into their functional constituent tracts—resolved by the distinctions effected by processes of disease and subsequently by those of development. But meanwhile another process of analysis was taking place. This process was one of extreme importance

and extreme interest. It rested partly upon direct observation and partly upon inductions of a theoretical nature founded on these observations; and strange to say, even the observations have been to a large extent lost sight of. They were the researches of the elder Remak, of Max Schultze, corroborated by the scarcely less important investigations of our own Beale. Before then, and indeed since then, the axis cylinder of a nerve fibre had been thought of as a unit, as a solid conducting substance comparable to a conducting wire through which a current of electricity passes, and a bundle of nerves has been thought of as analogous to a submarine electrical cable. But the number of separate units capable of isolated conduction contained in such a structure is wholly incommensurate with the needs, motor or sensory, of the parts to which the nerve goes. So incommensurate, indeed, are the nerve fibres for their various and different functions that it has led one of the latest writers on the subject to deny the possibility of demonstrated facts because the nerves fail to furnish a sufficient number of separate elements to subserve the separate and distinct functions that can be traced. The marvellous discrimination of sensation in the skin, the way in which touch, heat, cold, and pain are distinguished in the most minute areas of the integument is one of the most remarkable and most mysterious phenomena of the nervous system; and it must be confessed that we are unable to discern adequate mechanisms for this function if we regard the axis cylinders as separate structures capable only of separate function. But thirty years ago the researches of the investigators to whom I have referred established a fact which has been, like so many other facts, lately rediscovered. They observed that the axis cylinders of nerve fibres are not solid concrete objects; they observed that these axis cylinders consist really of a number of separate fibrillæ not only separate but having a distinct course through the nerve cells, and probably isolated to such an extent as to be capable of separate conduction, so that each constitutes a path having a distinct termination, distinct in the periphery and distinct in the centre, and that every nerve fibre may not improbably represent a structure as complex, as complicated, as the whole nerve, but of equal capacity for elaborate and even multiple function. The division of the axis cylinders at the periphery and in the centre doubtless consists in the separation of the fibrillæ.

However great may be the difficulty in discovering the needed



room for the function which undoubtedly exists in nerve elements as generally conceived, this difficulty disappears before the conception which I have described. It is marvellous in its multiplicity, but it is not more marvellous than the multiplicity of function, and we must not hesitate to grasp a correspondence of structural arrangement with functional needs. Yet this idea leaves us only on the threshold of the analysis which we must boldly face if we are to perceive our therapeutic needs and the manner in which these must be met. The conception of the fibrillar structure of the axis cylinder is a matter of direct observation, and capable of actual depiction. Beyond it the eye of reason must guide us, for actual observation leaves us far from the region in which the chemical processes take place with which we are concerned, and in these alone can we look for the processes which subserve function, for the beginnings of disease and for the beginnings of such restoration to health as we can achieve by means of treatment. In what manner the fibrillæ are made up of these molecules we cannot even conjecture, any more than we can conjecture the manner in which the molecules are made up of the atoms of which they undoubtedly consist. But here we have an organic laboratory in which nerve energy is being for ever evolved, in ordered or disordered form ; here we have a laboratory in which the atomic processes are at work unceasingly, with the results which we feel and see, although we cannot trace and cannot even guess the path by which they are attained. As I have said, it is in these molecules that we must look for the chemical processes by which the nerve energy is produced that manifests itself in what we term functional activity. The great fact for us is that the process, under the influence of life, is one of chemical nature. It brings the action of the nerve elements into the range of chemical phenomena, alike in health and in disease ; and it brings the old chemical therapeutics to our view, in a fresh aspect. We must not undervalue the non-chemical methods of treatment. Without doubt by their aid much is achieved, and much more will be achieved as the conditions of their use come to be better known and the indications for them better understood. But the problem to which I wish especially to direct your attention to-night is the value of chemical therapeutics and the rational foundation which can be discerned for it in the facts I have mentioned.

The first therapeutical principle to which I am anxious to direct



your attention, is the importance of a deliberate and careful application, to each practical problem, of every detail of the pathological facts that have been ascertained. Whenever you have to treat, or to endeavour to treat, a morbid process in the nervous system, before you consider what means you should adopt, you should strive to frame a mental picture of the process itself—an image of the changes that are taking place, molecular or massive; of the relations of the symptoms to the process which they manifest; and of the manner and degree in which these symptoms depend on either the primary process of the disease or on its secondary effects. Only when you have done this should you consider the practical problem of the best method of dealing with the changes which give rise to the symptoms. This may not seem a rapid process, and the need for it may not seem a sanguine doctrine; but few processes which relate to the therapeutics of diseases of the nervous system and can be regarded as approximately trustworthy, are safely rapid in execution. Still, the method is not altogether unhopeful, and each day's researches increase distinctly the prospect that success will attend its right application in a larger number of cases and that deliberate thought will prove more and more effective.

Kindly remember the words I used, "When the primary process of disease is molecular or massive." I had a special reason for thus using the term "primary." Whatever be the original morbid process, the symptoms themselves invariably depend on the minute changes, and it is most important to grasp the fact that these changes are often independent in their nature, and distinct from that of the primary disease. If the primary morbid process is a massive one, as a tumour of the brain, the symptoms which manifest its presence and declare its character are of minute nature, consisting in molecular alterations in the nerve elements similar to those which constitute the primary change in many diseases which have no massive cause. They are similar, for instance, even to the alterations in the diseases which we term "degenerative," which commence in the molecules themselves. The effects of a syphilitic lesion, such as disease of an artery causing softening, may differ very little from those of a degenerative disease, such as consists in the multiple alterations in the peripheral nerves in the limbs, or in the spinal cord, which are apt to supervene at a somewhat later stage and may constitute the malady we term "tabes."

How profoundly important this fact is in its therapeutical relations, how it explains the limitations to our power of treating each disease, I need hardly pause to point out to you.

The first great fact is that the source of nerve force is chemical energy, or rather it is the mysterious "tension," by which this chemical force is held latent and only released under certain conditions; but here we step into the region of that which is still too mysterious to be profitably discussed. Moreover, we cannot tell, we cannot even conjecture, what processes may be conjoined with that of the apparently simple transformation of the chemical energy into nerve force, by what these may be influenced, and to what they may be ultimately due. We must, therefore, leave a gap in the process of our thought and must not assume that the series of events which we perceive to be consecutive is really unbroken by others that we cannot discern, at least in any certain manner. But of the great fact of the source of nerve energy we may feel sure. It is established by too many corroborative chains of evidence to permit us to entertain any doubt of its validity, and it is of extreme importance for our present subject. If chemical force takes so important, not to say predominant, a part in the functions of the nervous system, it is not suprising to find that chemical substances, and the energy they bear, assume an equally prominent part in the production of the abnormal forms of action which we term "disease." The extent to which chemical agents generate disease was indeed scarcely suspected until the last few years, but during that time it has been amply demonstrated and amply confirmed. The example of it which is the best known and is at the same time perhaps the most typical in its cause and its characters is the multiple inflammation of the peripheral nerves which is produced by alcohol. Although this was thought at first to be peri-neuritic, to have its seat in the interstitial tissue and in the tissue of the nerve sheath, it is now abundantly proved by repeated observations that the nerve elements themselves are the structures which are primarily affected and that it must be upon these that the chemical agent is able to exert its influence. Analogous facts have been established with respect to arsenic and many other substances. We now see that chemical substances must be placed amongst the most important morbid agents capable of producing disease, and they produce disease, in point of fact, every day. The inflammations and degenerations, at any rate those which are



symmetrical and are produced through the agency of a blood state, are produced through chemical agency and through this alone. Note that unexpected results followed from this discovery. The perception of the nature of the acute specific diseases and of their dependence upon an "organised virus" entailed a corresponding application to the sequelæ of these diseases in the central nervous system. It was natural to refer these sequelæ to the influence of an agent of the same nature as that which caused the primary malady; but our eyes were to some extent blinded by an excess of light. When we perceived how potent was the capacity for producing disease possessed by the organisms which constituted the "virus," it was natural to ascribe to these not only the malady itself, but its sequelæ, and to lose sight of the relations of the consequences and indirect effects. It did not occur to us that it might not be correct to discard the older views so completely as we were disposed to do under the influence of the new theory. Men of science are strangely slow to learn the lesson which the swinging pendulum presents to them each day—the lesson of truth between extremes. They forget how seldom it is that old truths disappear before the new, although the scope of their application may be greatly reduced. In the case we are now dealing with, this has been true in a conspicuous degree, since chemical substances, due to the organisms, seem the chief cause of these sequelæ.

In considering the relation between the apparent cause and the apparent consequence, the first fact that forces itself on the observer's mind is the extreme irregularity of that relation, the great variation that is to be perceived in the time at which the effects on the nervous system result from the cause of the acute specific disease. On the general system the effects are produced within a certain limited time in the case both of acute and of sub-acute maladies, but on the nervous system the consequences are often long delayed—they may be delayed for weeks or months, or even for a year or more. It is difficult to give an explanation of this strange phenomenon except on the theory that it is the result of the action of a chemical agent and not of a living poison consisting of living organisms. Such a chemical agent may act on the nerve elements directly or indirectly, lowering their function or reducing their power of vital endurance, and in either case bringing about the same result at a near or distant period of time; but we



cannot refer such variations to the growth of the organisms themselves. A similar diversity, of similar significance, is presented by the great disparity in the intensity of the symptoms of the primary disease and of its late consequence. We can only understand this on the hypothesis that the connection is indirect and is of such a nature as that which I have indicated.

I might multiply analogous facts, but it is of more importance to ask, what do they teach, what lesson do they suggest to us? Note the salient features that we have remarked. Nerve force is in some way a product of chemical force, a result of the transformation of the chemical energy contained in the molecules that make up the nerve elements, and are themselves made up of the atoms whose existence we are compelled to assume. These atoms are derived from the material taken into the system, which by the chemical energy is conveyed to the molecules. The nature of the material taken in, thus determines the character of the energy which is liberated when the chemical processes occur between them and permit the transformation of the chemical force into nerve force. Thus the character of the latter depends on the processes of nutrition which are determined by the nature of the material taken into the system, and by its affinity for that which it already finds there. Some of the material may be inert—the atoms are incapable of effecting any change in the constitution of the molecules or in their function; some is suited to promote normal nutrition and to facilitate the liberation of energy in a normal manner; some degrades the processes of nutrition and leads to the abnormal action or imperfect action which we term “morbid,” and this may even induce, slowly or rapidly, the molecular decay that we term “degeneration”; lastly, if such abnormal nutrition already exists, we can perceive the possibility that it may be changed, that it may be augmented or lessened, by the influence of atoms thus introduced into the molecules, and that their function may be altered by the chemical processes which are thus induced. We seem to have, in these considerations, a rational basis for the use of drugs in the treatment of many diseases of the nervous system. The work of innumerable generations of practitioners, in their endeavour to treat these diseases, has consisted mainly or exclusively in the application to them of the influence that is to be obtained from such chemical compounds. It was from these that they sought to gain the power they needed, and their efforts were

believed by them not seldom to be crowned with success which, if not complete, at least was not to be disparaged. This method held its dominant position until the present generation, which has witnessed the advent of other methods, to some extent the rivals of the older one, to some extent its successors. The degree in which they can be regarded as its successful competitors is, however, a matter of opinion, but it is certain that they have led their advocates to disparage the method of treatment by the use of drugs to an extent which is at any rate large, and possibly unjustifiable. Under these circumstances it has seemed to me important to note the firm foundation which modern observation as well as ancient experience supply to the method in question. We have already, sufficiently for our purpose, glanced at the most important points connected with this evidence; but I have still to point out how pertinently the latest results of therapeutic science afford confirmation to the opinion and practice which have influenced so many of those whose wisdom we are perhaps rather too ready to doubt in the new light we have gained.

What is the latest result of therapeutic science? What is the latest product of therapeutic investigation? Can anything be adduced which deserves a place by the side of the tuberculin of Koch? It does not matter how such substances were discovered or are obtained. However important these conditions may be, they do not affect the fundamental fact underlying their manifestations, and on account of which I have thus dwelt upon them. The important fact for us is that they are chemical in nature and must produce their effects by the chemical processes into which they can enter and by the chemical force which they can evolve under certain peculiar conditions. The latest result of therapeutic science is thus at one with her earliest efforts: an action through a chemical agent by the mechanism of chemical energy.

The last point to which I would allude is one of a more personal nature, based upon personal experience. My own observations have given me, as regards certain classes of diseases, strong and increasing confirmation of the practice of our predecessors. The affections to which I refer are those which consist in and depend upon primary alterations in the molecular nutrition of the nerve elements, manifested by disturbance of their function, either transient or so persistent as to imply alterations of structure. These diseases include the maladies that we can most surely trace

in many cases to the influence of chemical agents. They also include others of similar features in which such causation is not traceable, but in which we may reasonably assume that a similar molecular process is manifested by them, although due to a different cause. These are the extensive class of affections that we term "functional," whilst the others include the equally extensive class of "nutritional" diseases. In both of them the beneficial influence of drugs appears to me to be beyond question. The agents employed need to be chosen with judgment, changed with discrimination, and used with perseverance—perseverance alike on the part of the patient and the practitioner. Given these conditions, I have been surprised at the amount of good that has been done in affections commonly looked upon as intractable—relief, arrest, and restoration. With each successive year's experience these results seem to me greater and more distinct, and to elicit more gratitude from the sufferers who are thus relieved. I regret that lack of time precludes any attempt to illustrate this statement, but my object is attained if I can succeed in bringing home to your minds the reasonableness of the experience, and in encouraging you to keep your grasp of the old when you seize the new.

---

*April 17th, 1893.*

## PYREXIA FOLLOWING THE ANÆMIA DUE TO HÆMORRHAGE.

By M. HANDFIELD-JONES, M.D. Lond.

Rises of temperature occurring soon after delivery always call for careful consideration, and necessarily give rise to much anxiety. It is therefore of the utmost importance that the practitioner of obstetric medicine should be thoroughly acquainted with all possible causes of such pyrexias, and should be able early to decide in what cases the symptom has a grave meaning and foretells the onset of septic intoxication, and in what cases the increased temperature is of trivial importance. The disturbing influence of lactation is well known, and rises in temperature due to this cause are constantly



recognised; many cases of pyrexia have been cured by a timely aperient, which has led to the evacuation of large, hard scybala: in vol. xxvi of the 'Lond. Obstet. Soc. Transactions,' Mr. Tait has done good service in calling attention to a series of cases in which puerperal pyrexia was due to various nervous causes. To-day our attention is directed to cases in which rises of temperature depend on the anæmia caused by acute losses of blood, such as may occur in instances of *ante-partum* or *post-partum* hæmorrhages.

In his admirable Lettsomian Lectures, delivered before the Medical Society in the year 1891, Dr. Stephen Mackenzie has fully recognised the frequent presence of a high temperature in cases of anæmia. Thus, in detailing the symptoms of the latter affection, he says occasional and irregular pyrexia is common in anæmia of whatever nature. I have found it in traumatic anæmia in previously healthy persons, and after hæmatemesis, and in my notes of cases of chlorosis I find slight degrees of pyrexia very common, "a mimic reproduction of the irregular *anæmic fever* that appears in pernicious anæmia" (Coupland). It is more pronounced in the latter than in other forms, but some cases of pernicious anæmia are throughout apyrexial. The fact that fever occurs in chlorosis is important, as its occurrence in pernicious anæmia, in concurrence with the exacerbations of other grave symptoms, has been regarded as evidence of blood destruction. Of pyrexia in pernicious anæmia the same author says, still more emphatically, pyrexia is present in the majority of cases. It is irregular in its course and duration. The temperature often runs up to 101° F. or 102° F., and may be even as high as 104° F. It does not remain high long. In the present communication I shall not attempt to deal with the subject of pyrexias occurring in the course of chronic anæmia, but shall concern myself only with those rises of temperature which are set up by sudden and severe losses of blood. I am the more disposed to confine my remarks within these limits, because little attention seems to have been directed to rises of temperature following acute hæmorrhages, though most practitioners are familiar with the occasional pyrexia of ordinary anæmia and chlorosis. In associating pyrexia with the anæmia due to acute hæmorrhage much careful investigation of each case is necessarily needed, for the causes of the bleeding, such as childbirth, injury, operation, &c., may in themselves induce conditions of inflammation or morbid change, such as would be naturally accom-

panied by rise of temperature. If the hæmorrhage and pyrexia follow childbirth, this care is especially necessary, since, owing to the unstable condition of the nervous system in pregnancy and parturition, the temperature condition is easily disturbed by such conditions as lactation, constipation, or mental emotion.

Great care has been taken in the selection of the following clinical records, so as to exclude, as far as possible, any case in which there might be a possibility that the increase of temperature depended on some concomitant morbid condition, and was not due solely to the anæmia.

CASE 1.—Mrs. S—— was delivered of her second child on the morning of March 9th, 1893, at 11.30. There was nothing noteworthy about the labour, which was fairly rapid and easy; but the third stage was attended with violent flooding. Indeed, the uterus was so toneless and so inclined to bleed, that it was not safe to relax one's hold of that organ for two hours after delivery. The same evening the temperature at 9 P.M. stood at 102·8 F.; the patient was very blanched, very thirsty, and complained of much headache. The pulse rate was 130. The accompanying chart shows that for some ten days after delivery the pyrexia was well marked. Throughout the puerperium the lochia, though scanty and pale, were otherwise perfectly normal, there was never any pain or tenderness about the abdomen, the breasts secreted freely and caused no trouble, and the bowels were easily regulated by small injections of glycerine. The pulse rate for a week kept above 100, and then slowly fell to 70. The patient had a very fair appetite, and made an easy and natural convalescence.

The notes of the following case have been kindly supplied to me by my colleague, Dr. Lees.

CASE 2.—Mrs. T——, primipara, was confined on March 21st, 1884, at 4.30 A.M. The labour was an easy one, and the placenta and membranes were expelled entire without difficulty. As, at the end of an hour, the womb was found to be well contracted, half a drachm of ergot was given to maintain retraction, and the patient was left. At 10 A.M. a rather severe hæmorrhage set in, but was checked by the injection of hot water and by pressure. At 2.30 P.M. the bleeding recommenced sharply, but was again checked; the patient, however, complained of feeling faint. A mixture of ergot and iron was ordered.

*March 22nd.*—Patient seems comfortable; next day the breasts began to secrete. Temperature was taken, and found to be 102·5° F. On the 24th and 25th headache was still troublesome, and the temperature had risen to 103·2° F. Quinine was now given freely, and in the evening the temperature had fallen to 101·2° F.; pulse 124. No shivering.

On March 27th, at 10.30 A.M., the following report was given: Bowels freely opened by senna. Headache still very bad. Sleep bad. Lochia normal, perfectly sweet; no marked tenderness anywhere over abdomen. Lung sounds healthy. Has a soft bruit over pulmonary artery. No bruit at apex, but first sound impure. Second sounds at base loudly accentuated. Urine normal; does not contain albumen.



Temp. 103·4° F. Pulse 140.

R Morph. gr.  $\frac{1}{4}$  + ammon. bromid. gr. x + sod. brom. gr. x; to be repeated in two hours.

Temp. 5 P.M. = 105° F.

Temp. 9 P.M. = 103·6° F. Pulse 123.

March 28th.—Mid-day temp. = 100·5° F. Pulse 107. Feels much better.

Temp. at 5 P.M. 102·4° F.

March 29th.—Morning temp. 100° F.; still some headache. Lochia quite normal. Plenty of milk secreted by breasts. Temp. 9 P.M. 102° F.

March 30th.—Better; slight headache. Temp. 100° F.

March 31st.—Temp. normal; child takes breast well.

CASE 3.—On January 8th, 1891, I was summoned by one of the midwives of the British Lying-in Hospital to see an out-patient, who had been confined on the morning of January 4th, 1891, and who had suffered with several attacks of severe secondary hæmorrhage since delivery. The temperature and pulse were reported to be considerably elevated. On arriving at the patient's house I found Mrs. N—— almost blanched by the repeated losses, and showing a pulse of 120 with a temperature of 102° F. Examination by the abdomen and vagina made it certain that the uterus, though spongy and poorly contracted, was not tender or in any morbid condition, the surrounding tissues, though lax, were healthy, and the discharges were perfectly sweet. There was a basal hæmic bruit, but no evidence of disease anywhere. The breasts were healthy, and the secretion of milk free. For nearly a week the pulse remained over 100, and the temperature fluctuated between 100° and 102·4° F., then pulse and temperature gradually fell to normal. The patient had been a weakly woman before delivery, and made a slow convalescence.

CASE 4.—Mrs. X—— had arranged to be attended from the maternity department of St. Mary's Hospital in her confinement, which was expected in March, 1890, but in February she was seized with severe *ante-partum* hæmorrhage, due to the partial separation of the placenta. The child was born dead, but the mother got over her labour safely. Five days after the confinement the junior resident obstetric officer sent for me, and asked me to see the woman with him. His story was that the patient, though pale from her great loss of blood, was doing well, but that the temperature would remain persistently high. The bowels had acted well. The breasts had been kept from lactating by pressure; and the lochia, though pale and scanty, were free from all fœtor. The abdomen was flat, the uterus seemed normal, and there was no tenderness anywhere. On examination I was able to confirm his statement, and except that the pulse was 118 and the temperature at 4 P.M. 102·8° F., I could find nothing wrong. From previous experience it was easy to assure him that the pyrexia and rapid pulse depended solely on the anæmia, and would shortly disappear. In this case the twelfth day was reached before pulse and temperature had reached the normal standard.

My colleague, Dr. John Phillips, has supplied me with notes of cases which illustrate the same point regarding the pyrexia after *post-partum* hæmorrhage. In one of his cases the lady was confined between 4 and 5 o'clock in the morning, and the expulsion



of the child was followed by severe *post-partum* flooding. On the evening of the same day the temperature was found to be standing at 104° F., with a rapid pulse rate. The pyrexia, however, slowly sank step by step with the pulse, and the patient made an easy convalescence.

It would be easy to bring forward notes of many similar cases, but the above are sufficient to afford examples, or rather illustrations, of the clinical point which is under consideration.

The cases so far quoted come entirely in the domain of midwifery, and it is in this department that I have noticed most instances of pyrexia, in connection with anæmia, but I have notes also of a few cases where the febrile condition was present without pregnancy having existed. At present I am anxious to gain information from my surgical friends as to the existence of these rises of temperature after traumatic hæmorrhages.

CASE 1.—Mrs. X.— was admitted to the new Boynton Ward, suffering from uterine prolapse; as she was troubled, at the same time, with a large crop of hæmorrhoids, I directed my house surgeon to operate, and remove the latter, before I commenced treatment of the uterine condition. The removal of the piles was successfully accomplished, but whether through slipping of a ligature, or insufficient torsion of some small artery, very severe bleeding came on the same evening, and not only was a large amount of blood lost *per anum*, but the rectum was found to be distended by large masses of blood clots. Next morning when I saw her, the anæmia was intense, the woman's mucous membranes being blanched. The temperature had fallen to 97°, and remained for some hours sub-normal; but on the evening of this day the temperature slowly rose, and for a full week it remained between 101° and 102° F., then it slowly fell again to normal. During the whole of this time the patient remained markedly anæmic, but had no bad symptoms, or signs of internal mischief. Ultimately she made a perfect recovery, and left the hospital fully restored.

CASE 2.—Mrs. S.— came under my care, complaining that for the last nine months she had been suffering with floodings at the poorly times. Her face was blanched, the breath was short on exertion, and she had all the signs of pronounced anæmia. Examination showed that the uterus was enlarged, the cervical canal patulous, and that a submucous fibroid existed. A few days later the cervix was dilated and the tumour removed, but in doing this a considerable amount of blood was lost, and the patient became much exhausted. The temperature, as in the previous case, fell below the normal for some hours after the operation, but then rose again, and remained for four or five days between 100° and 101·6°, though there was not the slightest evidence that any morbid condition existed in the pelvis to account for this rise of temperature.

CASE 3.—Case of curetting for fungous endometritis. I have on several occasions noticed when curetting to remove villous growths from anæmic

women that the temperature has been sent up, and remained up for some days, although the careful observance of antiseptic precautions, the absence of all tenderness and pain, and the perfect sweetness of the discharges have made it clear that no inflammatory or septic complication had been introduced.

In studying the subject before us, the thought at once strikes one, if pyrexia is the result of hæmorrhage, ought not the rise of temperature to be noted more frequently? But, as a matter of fact, it is only occasionally that one comes across instances of acute hæmorrhage in which some complications do not exist, or sequelæ follow. Thus, in cases of severe blood loss due to accident or injury any rise of temperature following the accident may be the natural outcome of inflammation set up in the damaged tissues. Again, in severe hæmorrhages from the lungs, or from the bowel, we have to consider the disturbing influence of such causes as tubercle or typhoid. Still more when dealing with the floodings of parturition the greatest care is needed, lest one should ascribe to the resulting anæmia a rise of temperature which really depends on a minor degree of lymphangitis, or on a slight amount of septic intoxication.

*Differential Diagnosis.*—Some points are brought into prominence by studying these cases of pyrexia: thus, in almost all the cases the hæmorrhage is followed by a short period of subnormal temperature, a period which may last only a few hours, or may be protracted from twenty-four to forty-eight hours. It rarely seems to overstep this latter limit. In many of the cases it was noted that the rise of temperature occurred within twelve hours after a severe bleeding, or, in other words, within so short a period that it was difficult to believe that septicæmia or inflammatory reaction could have any share in causing the pyrexia. Of course in cases following delivery, one is not forgetful of the exalted sensibility of the great nervous centres, and that emotion, fear, anger, and other similar causes lead to rise of temperature, but all such causes were undoubtedly absent in the cases under consideration.

There can be no question that the rises of temperature were most marked in women possessing a highly strung or nervous temperament, and I am well aware that I have seen cases in which the most severe hæmorrhage has occurred, and yet no rise of temperature has followed. These instances, however, have occurred in cases where the patients were of a phlegmatic or



lowly organised system. As regards the duration of the pyrexia, the average was about a week, the shortest time noted was three days, and in one case the return to the normal standard did not occur until the end of the thirteenth day.

*Causation of Pyrexia.*—It is difficult to assign with certainty a definite cause for the rise of temperature noted, but, taking into consideration the exhausted state of the nerve centres due to the loss of blood, the early period at which the rise occurs after the hæmorrhage, and the fact that the rise is most noted in women of a mobile temperament, it seems most reasonable to believe that the disturbance of the centres regulating the temperature is due to an induced condition of hyperæsthesia. An additional argument for this belief is found in the fact that the temperature falls *pari passu* with the decrease of the anæmia. Of course it has been suggested that the rise of temperature depends on the poisonous action of ptomaines, and that these latter, though constantly present, are only able to exercise their noxious influence when the resistant powers of the system have been lowered by some depressing cause, such as hæmorrhage. Judging, however, from the clinical records, it seems more reasonable to suppose that the pyrexia depends on an unstable condition of the nervous centres than on the presence of poisonous particles in the circulation.

## A FURTHER COMMUNICATION ON HÆMORRHAGE FROM ULCERATING BUBO OF THE GROIN.

By A. MARMADUKE SHEILD, M.B., F.R.C.S.

IN vol. x of the 'Medical Society's Transactions,' I narrated a case of this rare and dangerous complication of bubo of the groin. In that particular instance, a considerable aperture formed by ulceration in both the superficial femoral artery and vein. A false aneurysm had formed in the thigh, and I was compelled to ligate both vessels above and below the lesion. Gangrene did not ensue, but the patient ultimately succumbed to pyæmia. I was able to tabulate five published cases of this nature; the paper and the discussion demonstrated the general fatality of these cases. The present case differs from the former in many particulars, and is well worthy of relation as a sequence to it. Arterial bleeding



from abscess cavities is peculiarly dangerous and embarrassing. Any contribution to the literature of this subject must be of interest to the practical surgeon, and to those much engaged in the treatment of venereal disease.

On the 17th August, 1892, I saw a patient, aged 26, with a large "angry-looking" bubo in the left groin. Several glands were involved, the skin was dusky and congested, and indistinct fluctuation could be felt in several parts of the swelling, which was very painful. The cause of the glandular suppuration was a rupture of the frænum, which had now healed, but which had existed for a month, sore and neglected. The groin swelling had existed for about a fortnight. There was no sign of syphilis about this patient, and his health seemed good, except for the fever and distress associated with confined suppuration. He had been lately drinking freely, but not to excess. I advised that he should come to London, and have the swelling opened without delay, and this was done under ether on the 19th August. I found extensive undermining of the tissues. One recess ran towards the pubes, another towards the iliac crest, and a third downwards in front of the femoral sheath. These I laid open on the finger to their extreme ends, and as much disintegrated tissue as was loose was scraped away with a blunt spoon; some black, disintegrating blood clots were mingled with the sloughy tissue, and I could not but remark to the gentleman who gave the anæsthetic, that the case was one of some hazard from the likelihood of occurrence of hæmorrhage. No bleeding of any moment was evident at the time of operation, and the cavity was well dressed with iodoform and lint from the very bottom. All went well for the first three days. The patient was much relieved by the operation, but was restless and difficult to control. The sloughs had cleared out of the wound, which had been energetically cleaned and dressed, and now seemed to be healthily granulating. A small portion of the coats of the common femoral artery could be seen at one spot below.

On the 22nd August, the evening of the third day after the operation, I was hurriedly called to him on account of hæmorrhage. In spite of a firm spica, there had been enough arterial bleeding to soak through the pads and voluminous dressings, and to form some small clots in the bed. I at once removed all the dressings, and found the wound filled with clot. This was

sponged away, but no recurrence of bleeding took place, and it was impossible to see where the hæmorrhage came from, though the cavity was well sponged out. The main artery was beating forcibly, and a more extensive portion of its coats could plainly be seen in the lower angle of the wound. The cavity was again carefully packed with an iodoform compress; over this were laid several clean sponges, and finally a firm spica bandage, exerting considerable pressure. All went well for three days. I daily dressed the wound, washed it carefully out, and packed it with iodoform tampons, finally applying as firm a spica as the patient could bear. I was most anxious to keep the deep cavity in as near a condition to perfect asepticity as was possible, though from what I knew regarding these cases, my mind was full of grave apprehensions regarding it. On the night of the 25th August, about half-past 12, I received an urgent message to attend, and on my arrival I found that a very considerable arterial hæmorrhage was going on, despite the pressure exercised by the attendant nurses. I at once removed the dressings mingled with a quantity of clot, when an arterial jet about the size of a crow quill issued forth from the cavity. The jet of blood, though small, was very forcible, and its situation was over the inner aspect of the sheath and granulations covering the upper part of the common femoral artery. Digital pressure readily controlled it, but by no means could I secure the vessel, for pressure forceps, artery forceps, and the like, broke away as soon as applied, making the bleeding more evident and furious. Acu-pressure was, of course, contra-indicated, as the main vessel must have been included. Indeed I was, and am now, uncertain whether I had to deal with a hole in the main vessel, or with an ulcerated branch of some size close to the parent trunk. Considering the gravity of the question as to whether the common femoral should be tied above and below the bleeding point or further pressure employed, I determined to temporise, and here I had a good instance of the value of freely opening sloughy abscess cavities. The exact situation of the hæmorrhage was under control of the eye and finger, and no blood was extravasated into the tissues. Digital pressure was maintained until the morning. About 5 A.M. the bleeding was reduced to a mere trickle, so that pressure with a sponge controlled it, and rest for the fingers could be obtained. Some small sponges having been prepared and wrung out in iodoform emulsion, the wound



was quickly packed and strong pressure applied, which was ill borne by the patient. About 8 in the morning, Mr. Jacobson was good enough to see the case with me in consultation. The compresses were removed; no recurrence of bleeding took place, and the wound appeared healthy. Under these circumstances it was determined to give a further trial to pressure more methodically and severely applied than before, and Mr. Jacobson gave me his advice and assistance in its application.

The limb was firmly bandaged from the toes to the groin and padded with wool over the prominent points. A compress of iodoform lint was placed in the wound, and covered with several sponges of graduated size wrung out in strong carbolic lotion. This compress was kept in exact position by adhesive plaster, and a few spica folds of ordinary bandage. A long elastic webbing bandage was next applied as a spica, flattening and compressing the sponges upon the groin. Cotton wool was abundantly packed wherever the bandage pressed around the pelvis and under the thigh. This bandage was put on with much firmness, and in the aggregate, the amount of pressure in the groin must have been considerable.

Finally, a long splint was placed from the axilla to the foot, keeping the limb absolutely at rest. The discomfort experienced by the patient was considerable, and the pressure soon became almost unbearable, so he was kept heavily under the influence of morphia, and a careful watch maintained upon the condition of the foot and toes. The diet was reduced to slops to lower the force of the pulse, and all stimulants were interdicted. In this way, by the aid of very intelligent nursing and judicious morphia administration, pressure was maintained for eight days. No fever of moment occurred, and no pain which signified wrong action about the wound was manifest. The patient, however, several times complained of a severe shooting pain down the course of the artery, which has been observed before in similar cases.

On the eighth day I cautiously removed the pressure, and though there was free venous oozing from the depths of the wound, this soon stopped spontaneously, and the granulations seemed healthy. The cavity was irrigated for some time with warm carbolic lotion, dried with pledgets of wool, and again dressed from the bottom with iodoform and lint. Pressure was reapplied in the same manner, but not to the same extent of severity. In-



deed, already the elastic, though protected by pads, threatened to cause sloughs on the skin over the pelvis in several situations. After this the wound was dressed and daily irrigated with "red wash." Pressure was gradually relaxed, and discontinued altogether at the end of the third week. The wound granulated and contracted with rapidity, and the patient left for the country on September 29th, with the parts soundly healed. There was considerable muscular wasting from the disease and pressure, but no signs of aneurysmal bulging about the cicatrix.

In the paper before referred to I entered very fully into the nature of these cases, and the various treatments adopted. The terrible fatality of them and their great rarity were fully shown, both in the communication and by the remarks of subsequent speakers. Among the conclusions I drew on that occasion, was that pressure would not be very likely to succeed in cases of recurrent hæmorrhage. Sir William MacCormac considered pressure a very uncertain method of treatment, but Mr. Bryant referred to those cases where he had known it succeed. Mr. Cripps also spoke strongly of its efficacy.

In the present paper I wish especially to dwell upon the importance of a thorough trial of pressure if the parts allow, and if it can be borne and carried out over a sufficient time. A good deal hinges upon the condition, should the parts allow of pressure being employed. If extensive sloughing is going on, and the tissues are infiltrated and sodden with septic products, pressure is ill borne, and doubtless would seldom if ever succeed. The opposite condition, therefore, of a clean and healthy wound, without œdema and infiltration, is very essential if pressure is to have any success. Should there be an obvious breach of the walls of the main artery or vein or both, as in my previous case, attended with violent and persistent bleeding, ligature of the vessels would be a difficult but imperative duty. The case I have in this paper narrated, illustrates how difficult it is to be sure whether the bleeding comes from the main vessel or from a considerable branch ulcerated close to the parent trunk. Exactly the same difficulty has occurred to surgeons in cases of punctured wounds in the vicinity of the great arterial trunks. In these cases it has more than once happened that after the main vessel has been exposed by a tedious and difficult operation it has been found intact, the real source of the hæmorrhage remaining doubtful. Pressure judiciously employed

in such cases has, as pointed out by Mr. Cripps, been frequently successful, and should always receive a thorough trial in cases of doubt. I believe that in the instance related, a branch, possibly the circumflex iliac, had sloughed close to the common femoral, and though the parts were too rotten to bear the ligature or other material methods of checking hæmorrhage, yet a sufficient length of vessel remained to become sealed by clot after long continuous pressure.

I would venture also to insist upon the great importance of very freely opening sloughing abscesses about the great vessels. In the present instance had an incomplete opening been made, the blood would have infiltrated the tissues, which would have also been foul and septic, and pressure would probably have been out of the question. The practice of treating cases of this sort with powerful astringents, especially iron, is also very questionable. Inflammation and sloughing are intensified, and should the bleeding not be checked the damage done to the soft parts is considerable, and very prejudicial to the success of any further measures. The actual cautery is open to the same objections.

In conclusion, I trust that this case and paper may prove a small but not unimportant contribution to the literature of this subject and the perilous complication of ulcerating bubo of the groin. The accident is so peculiarly embarrassing from a surgical point of view, that all cases deserve our careful notice and study. It is indeed singular, that surgeons of life-long experience in the treatment of venereal buboes, should have never met with these cases, and yet that two of them should have occurred in my practice.

This only exemplifies the common remark that rare cases run together, and leads me to hope that as I have been peculiarly fortunate or unfortunate in my experience of this distressing complication of ulcerating bubo, I may never again be called upon to contend with it.

Mr. LOCKWOOD remarked that there were two conditions requiring treatment—the bleeding from the ulcerated point in the artery and the condition which produced the ulceration. As the artery was injured by an active process of ulceration it was often impossible to ligature the bleeding vessel, and the only alternative was to freely expose the bleeding point and apply pressure. For sloughing ulceration the treatment by continuous immersion in hot water was very good, especially when coupled with the after-application of strong solutions of tartrate of iron, which was a better application than iodoform.

Mr. SHEILD, in reply, said that in the case related in his first paper

the hæmorrhage occurred before the bubo was opened. He agreed as to the value of the warm bath treatment; and he mentioned a similar case which had occurred in a large provincial hospital in which amputation at the hip-joint was ultimately performed, but the patient did not recover.

---

*April 24th, 1893.*

## ON THE DIAGNOSTIC SIGNIFICANCE OF HÆMOPTYSIS IN AORTIC ANEURYSM.

By T. GILBART SMITH, M.D., F.R.C.P.

MR. PRESIDENT AND GENTLEMEN,—In the few remarks that I have the honour to bring under your notice, I desire to elicit the opinions of Fellows of the Society, whose knowledge and experience in the matter are greater than my own, rather than weary the Society with a lengthy paper—knowing well that the discussions which take place in this room are not the least valuable portion of the work of our Society.

The consideration of the part played by hæmoptysis in the symptomatology of aortic aneurysm is not unimportant, seeing that in a large number of cases of the disease which I have had under treatment, hæmoptysis has been present, and, in some of these cases, the spitting of blood has been the first symptom complained of, and the first symptom which led to the recognition of the disease.

Hæmorrhage in the form of hæmoptysis due to aortic aneurysm is of two kinds: (1) *Direct*, from leakage from the sac; (2) *indirect*, caused by pressure. That *direct* hæmorrhage need not necessarily be immediately fatal there is ample evidence, most of us having, doubtless, seen such cases in which the ulcerating opening of pin-hole character is blocked by coagulation, which for a time—short, rather than long—resists the pressure behind and staves off the evil day.

It is of the *second* form—*indirect* hæmoptysis due to pressure—that I would refer. This form is referable to *four* causes:—(1) Pressure on the air passages; (2) pressure on the blood



vessels; (3) pressure on the lung substance; and (4) interference with lung circulation owing to secondary cardiac valvular incompetence. Pressure on the air passage induces a local congestion of the lining membrane of the tube, which, in its incipient stage, by the irritable and often paroxysmal cough and scanty bronchial secretion slightly tinged with blood, heralds the oncoming of a more advanced stenosis accompanied by all the evidences of resulting bronchitis, emphysema, and well-known lung alteration due to narrowing of the trachea or bronchus.

Pressure on the blood vessels was well illustrated in a case of aneurysm where a sac budding off from the ascending portion pressed upon the right pulmonary artery, and almost occluded it, resulting in chronic inflammation and hæmorrhagic infarction of the left lung.

Hæmoptysis due to valvular defect—the result of aneurysm—was in evidence in another case where a diffused aneurysmal dilation of the aorta led to regurgitation through a stretched aortic ring with healthy valves, and to the intense suppuration of both lungs with venous blood. A short time ago a strong, florid, well-built, middle-aged man was admitted under my care at the London Hospital, suffering from what seemed to be capillary bronchitis with marked dyspnœa. Loud rhonchi and crepitant rales were everywhere audible over the chest, and masked the cardiac sounds. His expectoration was bronchial and tinged with blood. Shortly after admission, I was informed that he was bringing up pure blood, but there was some discrepancy of opinion as to whether it was vomited or expectorated. On cross-examination, however, I found it was both, which led me to suspect that the general bronchitis resulted from pressure of an aneurysmal sac upon the trachea, with a leakage into it or into the œsophagus. On the night following my visit a rush of arterial blood closed the scene; and, in due course, a small sac was found with a perforation communicating with a compressed trachea, and also an almost successful attempt at like communication with the œsophagus. In this case there were no other pressure signs than those mentioned, and no stridor, tremor, or pulsation pointed to aneurysm. The direct hæmorrhage from the bursting of the sac did not at once prove fatal, and was, doubtless, preceded by indirect hæmoptysis due to the induced bronchial congestion. In another case, where the signs of aneurysm were scanty, there was hæmoptysis from a

portion of lung carnified by direct pressure of the super-lying sac.

A man now under observation at the London Hospital presents all the evidences of aneurysm with definite pressure signs on the left bronchus with laryngeal affection. When, some two years ago, he was first seen he was *minus* these advanced signs, and simply complained of anginal symptoms. With him, the advance of the disease and its more marked signs have been announced by bronchial expectoration tinged with blood.

It seems to me, therefore, from the consideration of these and many other similar cases which we have all seen, that the occurrence of slight hæmoptysis is not without significance in certain ill-defined cases. Given a patient, perhaps with a syphilitic history, all the more likely if he has the physique and appearance such as we find generally in the victims of this disease; given symptoms of uneasiness, if not of distinct pain, in the chest of a spasmodic character with few signs of a cardiac nature other than perhaps those of high cerebral tension, and presenting no symptoms pointing to pulmonary disease beyond a paroxysmal cough with but little expectoration—and that tinged with blood—and when further, if in such a case we can, after careful examination, eliminate ordinary disease of the lungs, malignant growth, kidney and heart disease, I am of opinion that in such a case we have just reason for suspecting the existence of thoracic tumour; probably aneurysm, and for anticipating at no distant date the fuller development of more definite symptoms of increasing pressure, together with the tangible signs of the correctness of our forecast.

The PRESIDENT agreed generally with the views expressed in the paper, and could quote many cases to confirm the truth of them; but he thought that mediastinal tumours, in their early stage, were attended with all the symptoms that might characterise aneurysm, except a rush of blood after rupture. He referred to instances showing that rupture of an aneurysm was not necessarily immediately fatal.

---

*May 1st, 1893.*

## THE ANNUAL ORATION—PHYSIC AND LETTERS.

By WILLIAM MITCHELL BANKS, M.D. EDIN., F.R.C.S. ENG.

MR. PRESIDENT AND GENTLEMEN,—I must, in the first place, thank you for the great honour you have done me in asking me to speak here to-night; although it is truly no easy task to address the Medical Society of London in the room which has echoed the wise and eloquent words of Marshall Hall and Humphry, of Hutchinson and Lister and Crichton Browne. That my words can never compare with theirs I know full well, and so I pray that this audience to-night may be merciful and long-suffering towards me. The best thing a man can talk about is that which he has most recently and most earnestly been thinking about. I have said—indeed, I am vain enough to hope that I have done—some things for the furtherance of medical education; and, in the course of considering what professional training was best suited for a youth so as to make him a well-educated medical practitioner, I have been tempted to go further and to inquire into the position of the said practitioner as a well-educated man. And so to-night, as I am free to choose my subject, I will leave alone strictly professional topics (for these we have always with us) and ask you to take a glance at the relations between our profession and the world of letters. Are the study and practice of medicine of a nature to attract men to the field of literature? I fear not. Our studies, although almost entrancing in many of their departments, give no play to the imagination. The branches of the femoral artery and the sciatic nerve are, to all intents and purposes, numbered, fixed, and immutable. When we are trying to unravel the functions of brain or muscle or gland there must be no rein given to speculative hypotheses—sweet as stolen fruit; there must be no poetic “fancy free” to dazzle and divert our sober reason. The light of science is given forth from the clear, steady-burning lamp of the student, and not from the fiery cresset that flares amidst shipwreck and storm or lightens the skies on



nights of glorious and bloody battle. And when we come to the actual practice of our art, what are we confronted with? With sickness, with pain, with grief, with misery, with death—with so many things that are sad and repulsive that many a time we have to string up all our inner man to battle against them. It is often no easy task for the medical man to maintain that amount of cheerfulness without which his own life would be wretched, without which his patients would be bereft alike of hope and comfort. The temperament so engendered is certainly not that which will be likely to direct a man's mental footsteps down the sweet, shady alleys of poetry or fiction; and yet, in spite of all that, of the three learned professions—divinity, law, and medicine—I believe we stand foremost in the number of distinguished literary men who began life in our ranks. Need one mention Goldsmith? “The Deserted Village,” that sweetest poem in our language, and ‘The Vicar of Wakefield’ will be read whilst an English tongue wags in an English head. And there was Akenside—forgotten as physician to St. Thomas's Hospital, unknown as Gulstonian Lecturer and Fellow of the Royal Society, but still remembered and admired as the author of ‘The Pleasures of Imagination’ and of some of the most beautiful pastoral poems imaginable. Sir Samuel Garth, the life-long friend of Pope—the man by whose kindly help the remains of Dryden were borne to their last resting-place—was an admirable physician and no bad poet either. “The Dispensary” sounds a curious title for a poem; but Dr. Johnson said “it was on the side of charity against the intrigues of interest, and was, therefore, naturally favoured by those who read and can judge of poetry.” We can claim old Crabbe, too—“Nature's sternest painter, yet the best”—for he was a general practitioner before he became the Duke of Rutland's chaplain. I am delighted to find Crabbe coming again into public favour. Modern poetry is rather like some kinds of modern painting; there is so much of the symphonist and the impressionist about it that it passes the wit of the average man to make head or tail of it; but Crabbe's work is like a picture by old Teniers or Van Mieris. It tells its own tale, and its colours are as clear and brilliant as on the day they were laid on the canvas; and if we pass from poetry to prose the name of Smollett is no bad name to conjure with. Day by day a cascade of novels tumbles on the heads of the British public, drenching them with words,

words, words. How many of these have the stuff in them to last a hundred and fifty years and still be fresh and racy, like ‘Roderick Random,’ and ‘Peregrine Pickle,’ and ‘Humphry Clinker’? And then there was the handsome, courtly, mirth-loving Arbuthnot, Queen Anne’s physician, with whom Swift used to prowl about St. James’s arm in arm, and whom he used to talk about in the “Journal to Stella.” What fun there is in ‘Martinus Scriblerus,’ whilst from the ‘History of John Bull’ dates the origin of the conventional Englishmen whom ‘Punch’ draws for us now. Pope sadly but gratefully said of him—

“The muse but served to ease some friend, not wife,  
To help me through this long disease, my life  
To second, Arbuthnot! thy art and care  
And teach the being you preserved to bear,

\*            \*            \*            \*            \*

Oh, friend! may each domestic bliss be thine.”

It has been truly said that the name of John Locke is as little associated with the profession of medicine as that of Sir James Mackintosh, who was a practising physician till ambition and poverty made him select a more lucrative vocation and turn his energies to the Bar; but that the great philosopher actually practised medicine at Oxford there is no doubt. Sydenham relates how his method of treating acute diseases had received the approbation of Mr. John Locke, “who had examined it at the bottom, and who, if we consider his genius and penetration and exact judgment, has scarcely any superior and few equals now living.” The present is the day of mental exisceration, the day of sentimentalists, who agonise in public and dissect their emotions at so much a paragraph. What a contrast to read the ‘Religio Medici’ of old Sir Thomas Browne, and learn from his simple and manly words the secret of the faith that was in him.

If we come nearer to our own time, there was Mason Good, who translated ‘Lucretius’ whilst tramping the streets of London to visit his patients, and Leyden, the great Oriental scholar, who mastered nearly all the languages of the East, and gave to the English readers the ‘Commentaries of Baber;’ and there was Dr. Thomas Young, who first deciphered the writings of the old Egyptians, who called to life the dead history of a bygone world, and who summoned the mummy of an ancient king to tell the story of 3,000 years ago; and ‘Delta’ Moir, the country doctor



who worked amongst the fisherfolk and colliers of Musselburgh, and adorned the pages of 'Blackwood.' Even now we are not without a representative in the world of letters, for have we not still with us, aged in body but alert in mind, the "Autocrat of the Breakfast Table" ?

In scholarship, too, amongst the older heroes of our craft we are proud to reckon some giants. Linacre, who founded the College of Physicians and was its first President, was probably one of the most learned men of his time. He was the first Englishman who read Aristotle and Galen in the original Greek. I fancy the last must be dead. At any rate, I never met him. Linacre actually taught Greek in the University of Oxford. Both as a scholar and as a physician he ranked so high that Henry VII placed him near himself and entrusted him with the education and health of his son Prince Arthur. Just imagine living in the house of Lorenzo de Medici, teaching Sir Thomas More, and having Erasmus for your friend and patient, as he had. And what a famous successor did Linacre have in John Key. Associated with letters not less than with medicine, he raised on the foundation of Gonville Hall that college in the University of Cambridge which bears his name, and in whose chapel lie his bones, with the simple epitaph "Caius fui." In that delightful book, 'The Gold-headed Cane,' there is a charming scene, where Radcliffe visits Mead in his library. He says: "As I have grown older, every year of my life has convinced me more and more of the value of the education of the scholar and the gentleman to the thorough-bred physician. Perhaps your friend there (pointing to a volume of Celsus) expresses my meaning better than I can myself when he says that the discipline of the mind—*quamvis non faciat medicum, aptiorem tamen medicinæ reddit*—though it cannot make a man a doctor, it makes him all the better doctor. This was surely a noteworthy saying of a man whose professional life was so successful that his money built the Radcliffe Library, Observatory, and Infirmary at Oxford, and founded the Radcliffe Travelling Scholarships. And it is the more noteworthy because Radcliffe was not much of a scholar himself, insomuch that Sir Samuel Garth said a very funny, if rather severe, thing about him, viz., that the notion of Radcliffe bequeathing his fortune to endow a library was as if a eunuch were to leave his money to build a harem. However, gentlemen, when we pay a visit to Oxford or Cambridge,



we may cock our beavers as we walk past the Radcliffe Library or Caius College, and remember that two doctors built them. Mead, again, who succeeded Radcliffe as President of the College of Physicians, was a most accomplished scholar, and one of the most erudite men of his day. There were few such patrons of learning even amongst the nobility. What meetings those must have been with Pope and Newton and Bentley in his great library, with its ten thousand books, its marble statues of Greek philosophers and Roman emperors, its busts of great English poets, its collections of bronzes, gems, intaglios, Etruscan vases and coins. He made a magnificent income, and he spent it magnificently. After his death it was said of him that of all the physicians who had ever flourished he gained the most, spent the most, and enjoyed during his life the highest reputation, not only in England but abroad.

To come down from the intellectual giants of old days to our own time, I think we must frankly admit that the enormous amount of professional knowledge which the modern practitioner of medicine has to acquire effectually prevents him from rivalling his predecessor of old in the field of general literature. To attempt that would involve raising the sum of our lives from the threescore and ten years to the century—a thing, by the way, which I firmly believe will come to pass; but, whilst admitting this, have we not been neglecting too much that general learning and varied knowledge (apart from things medical) which are understood to characterise the members of a profession which proudly calls itself a “liberal” one? I fear we have. There never was a time when our calling was held in such high esteem by our fellow-men as now. Never before was the individual medical man so highly trained and so thoroughly versed in his own special work; but if you ask me what I think of the accomplishments, the mental culture, and the extent of reading of our profession as a body, I am bound regretfully to say that they are not as extensive, not as high, as they ought to be. In looking into the reasons for this state of matters, it appears to me that three things conspire to account for it. Firstly, there is the very defective school training which boys destined for our profession for the most part go through; secondly, there is the fact that so soon as medical study commences, there comes an interval of five years so enthralling in its claims on the student’s brains and time that it is an absolute

blank to him as regards general reading ; during that time he loses the habit and the love of it ; and, finally, in after life there is the exacting and exhausting nature of our occupation, which too often drags the medical man down to the level of a mere patient-visiting machine.

First, then, let us take up the question of inferior school training. This especially exists in England. The youths who elect to join our profession are drawn from the middle class of society. I would even go further and say that only a certain proportion of them are drawn from the upper middle class. Now, the great majority of these youths have received their education as boys in the private preparatory day schools of their towns, and I venture to say that in the whole range of educational institutions, from the highest to the lowest, there are none where teaching is so bad as in this class of school. That very sensible people, the Germans, do not believe in inspired schoolmasters ; they believe in the science of pedagogy, and in the business of teaching the teacher how to teach. The Scotch are credited with being a money-loving people, but it must in fairness be admitted that they have loved learning as well. Now, as far back as I can remember, there was in Edinburgh a certain "normal school," as it was termed, where young men and women were trained to be teachers. The truth is, that at the present moment the middle-class boy has not even the guarantee for good teaching that the School Board boy has ; for not only are the teachers of the latter most carefully prepared for the business of their lives, but the manner of their teaching and the quality thereof are subject to constant and careful inspection. From the lower middle-class day-schools of our great towns I assert that at present we get only an inferior product. I often ask a new dresser to read a report of his case and at the end of it am compelled to say, "Sir, your production is on a par with the letter of the cook to her intimate friend, which winds up with 'this cums hopping.' " In truth, to that young gentleman grammar, capital letters, and stops are of no more moment than vowels to an etymologist. As for writing out a prescription in full, the Latin gender and genitive case present such insuperable difficulties that Pot. Iod. is about as far as we generally get. Now, it is clear that two persons are to blame in this matter. The first is the teacher, who does not teach the boy thoroughly ; the second is the examiner, who does not examine the young man



thoroughly. There are certain university entrance examinations in arts for medical students which exact a good amount of knowledge from the candidates; but, possessing, as I do, a very intimate acquaintance with the subject, I maintain that with regard to the examinations for the licensing colleges there are a vast number of students rubbed through their sieve who ought to have been caught and retained on the way. As to these colleges publishing their subjects and their questions, and offering them to public view as evidence of the high standard of their examinations, I value their printed papers at the value of the paper they are printed on. What I want to see is the answers to their questions; but these are not forthcoming. The General Medical Council, in exacting a five years' course of medical study, has done a very remarkable thing for the advancement of our profession; but with a uniform five years' curriculum the method of spending the five years will soon shake itself into shape and therefore the question of the strictly professional studies of the student need not for a long time occupy much thought. What is now wanted is that the Council should look more stringently into the entrance examinations. Let them get hold, not of the question paper of the examiner, but of the answer paper of the examinee, and let them ascertain what the standard really is which passes a man through the portals of our profession.

There is another matter which is telling seriously upon the sound education of our boys, and that is the undue prominence given in the present day to athletics. Now, let me premise by saying that my age and proportions at the present day are hardly such as to justify me calling myself an athlete, nevertheless I have all my life been a lover of sport and exercise. I have the profoundest belief in the *corpus sanum*. I know that it is the outdoor life and habits of Englishmen that have enabled them to fight and conquer and colonise in every climate of the world, from Hudson's Bay to Ceylon; but it seems nowadays to be forgotten that, after all, the *corpus sanum* is merely the fleshly tabernacle of the *mens sana*. I object strenuously to a boy being encouraged to regard the life of a mere athlete as the highest to which a human being with an intellect can aspire; but at English schools thews and sinews seem to be the only objects of admiration and respect. Brains are out of fashion. Well might the German schoolmaster say that the English boy plays at his work and works at his play.



I have asked many a proud mother about her son at some public school and been told with a radiant air, "Oh, he is doing splendidly, getting on *so* well." This means that he is captain of the school eleven, or has got into the first football team, or is stroke of the school boat. Never by any chance do I hear of the boy's position in his class or form, or of his progress in his studies, or of the prizes he has gained. In fact, the studious boy is regarded with contempt by the great majority of his fellows. The very masters have to be athletes. One knows the usual style of advertisement for a junior master: "Must be Church of England and a good cricketer," like the crack emigrant ship of former days which was advertised to carry a cow and an experienced practitioner. I assure you, gentlemen, so little have our students been trained to use their brains as schoolboys that it takes most of them, after they have scraped through an easy preliminary examination, the best part of a couple of years to get into habits of steady, methodical, hard work. As for the fine, handsome young gladiators turned out by Eton and Harrow and Rugby, the School Board boy is ousting them out of the office and the counting-house, whilst getting into the army nowadays means work. Too often their expensive education is found only to have fitted them to go farming in Manitoba, cattle-ranching in Texas, sheep-raising in New Zealand, or bar-tending in Australia. The worship of bone and muscle has gone a shade too far in this country. I trust the pendulum has swung to its furthest. In attacking the schools where our students too often receive their early training, I would have you remember that it is not so much the things that are taught as the manner in which they are taught that I am criticising. What I complain of is a want of sound, honest, thorough "grounding" in a few things, and an omnipresence of superficial smatterings. Also of a failure to impress the boy with the importance of the fact that upon his early work very generally hangs his whole future life; likewise of a tendency to pay attention solely to those boys who evince a natural aptitude and liking for work, whilst the rank and file, who do not like work, are not whipped up and drilled into it until they do like it. As to what a schoolboy should learn, *tot hominibus quot sententiæ*; but I have lived sufficiently long to have seen and watched a great many schoolboys grow into men. As a result, I will propound what will doubtless seem to you almost a paradox—viz., that if a boy

destined for a profession were to be taught nothing but reading, writing, and arithmetic till he was 11, and after that nothing but Greek and Latin and mathematics till he was 18, at 50 that boy would turn out a more widely cultured, better-read man than if, in his early years, he had been stuffed with geography, history, philosophy, and the twopenny-halfpenny fragments of chemistry, botany, and zoology which constitute school science. All these things will come to him in time. Let him learn thoroughly the elements and the rudiments.

Let us now turn to point number two—the blank in the medical student's literary life caused by his curriculum of scientific study. At a time when other young men going into professions are reveling in a delightful freedom from schoolboy drudgery and wandering at their own sweet wills down the nearest and most fascinating glades of literature, the medical student must sit down to five long years of hard and incessant work at science. During that period there is no possibility whatever of his keeping up his general reading. Every hour must be given up to the inexorable necessities of his professional work. Before I began to study medicine I was an omnivorous reader; but so completely did my medical reading crush out of me all desire for general literature, that it was long after I graduated ere the desire for it returned. I had almost to force myself to it. Indeed, had I not been thrown into the society of some professional friends, who were men of cultivated tastes and wide knowledge, the old love might never have come back. On the other hand, had I been plunged into a hard, busy, general practice, or planted away in a lonely country district, the local newspaper and the weekly medical journal might well have limited my mental horizon. Not for a moment do I mean to say that the varied studies involved in a medical education are not pleasant. They are more than that—they are entrancing. Every day brings fresh delights—the delights that children experience who ramble on a holiday into some unknown forest. But when they are over, what then? Then comes practice, with the weary, harassing, often disappointing, realities of life. Can the practitioner fly for solace and repose for his jaded mind to his student text-books? I trow not. The essay, the review, the poem, the incident of travel, the glamour of history, the romance: these are the things that for a short, sweet evening hour or two will carry him into a land where there are no querulous complainings of sick



men, no tearful faces of anxious relatives, no thankless words of ungrateful patients.

This brings us to our third head—the argument that, when the practitioner comes home jaded and exhausted by a day of toil, he has neither bodily nor mental strength to sit down to read; but my contention is that, if the practitioner has learned to love good books, he will find in them the very thing he needs—rest and comfort such as he cannot get from anything else. If he has lost the love of letters he must force himself back to it. There may—there will be—an effort at first. Nothing that is really excellent is easy to do or to find. It is as with a beginner practising scales on a musical instrument—weary, monotonous drudgery. But after he has patiently mastered these difficulties, he suddenly makes the discovery that he can play tunes, and straightway the whole realm of music lies open to him. Ever after he flies for his chiefest delight to that instrument which at first was so ungrateful and irksome. I would say, moreover, to those who hold that the life of the average practitioner is so engrossing and fatiguing that the utmost he can do is to read his newspaper and his medical magazine that there is no necessity to go outside the limits of our own profession to see how erroneous that view is. Look at the men who are our acknowledged leaders. They are, every one of them, men of cultivated minds, who know many things outside their own special craft. Surely their lives have not been easy ones: lives devoted to hard professional toil, to laborious scientific research, to the strain of lecturing and teaching. But there is none among them who has not found time to put into the prescription of his daily life what the old apothecaries used to call the “corrective and adjuvant,” a good book by the fireside at night. Every wise man feels in his heart that, if he lives sufficiently long, a time will come when ambitious toil and money-getting labour will cease to satisfy. He recognises early the fact that in order to enjoy his old age he must not only have well-lined pockets but a well-stored brain; for the Frenchman La Bruyère was right when he said that man too often employs the greater part of his life only to make the remainder miserable.

Ours is, no doubt, a hard and trying occupation; our climate for the most part is dull and depressing, our big towns are gloomy and unlovely, and we are said to take even our very pleasures sadly. Heinrich Heine, who never lost an opportunity of venting



his spleen on England, nevertheless worshipped Shakespeare. He called him "The Sun of England." He said: "Shakespeare has, indeed, been a spiritual sun for that country, where the real sun is wanting twelve months in the year; for that island of damnation, that Botany Bay without a southern climate, that stone-coal-stinking, machinery-buzzing, church-going, and vilely drunken England." For his charming comparison of the greatest of poets to the sun that lightens up the Englishman's home we may forgive Heine his other savage words. Surely, with Shakespeare and Byron and Tennyson, with Scott and Dickens and Thackeray, with Carlyle and Macaulay and Prescott, even the humblest practitioner should be able to draw down some sun into his soul. Sir John Lubbock, in his own charming way, has always tried to make us look upon our books as our friends. They are, in truth, a man's very best or very worst friends, just as he chooses to use them. There are not many men who intentionally cultivate the society of blackguards, and so there are not many who of *malice prepense* set themselves deliberately to read filthy or degrading books. But there are a great many persons who are fond of pleasant, attractive, careless friends—friends who are not too particular about anything; who do not do anything very bad, but who certainly do very little good; whose conversation, though amusing, is trivial, and who at heart are untrustworthy and unsatisfying. It seems to me that in literature that sort of friend corresponds to newspapers, trashy novels, and cheap magazines—all pleasant acquaintances in their way, but not good enough to be made the companions of a lifetime. Once a man elects to ally himself with these, his literary decadence is ensured. *Noscitur a sociis*. If you find him constantly in their company you will look round in vain for his bookcase. It is an immense pride and pleasure to us to think that of all the learned professions none in late years has advanced—none is advancing—in public estimation so rapidly as ours. Medicine, in place of being the scoff of every would-be wit or poetaster, as it was two centuries ago, is now regarded with astonishment and respect. We are not merely healers of the individual units of the population whom we call our patients. All through the length and breadth of the land medical officers of health stand on guard to ward off infection and pestilence from whole districts and cities. In the year 1872, at Manchester, that most sagacious and prescient man, Lord Beaconsfield, spoke

these words : "In my mind the great social question which should engage the attention of statesmen is the health of the people, for it refers to all those subjects which, if properly treated, may advance the comfort and happiness of man. A very great man and a very great scholar two or three hundred years ago said that he always thought that in the Vulgate that wise and witty King of Israel, when he said *Vanitas vanitatum, omnia vanitas*, should really have said *Sanitas sanitatum, omnia sanitas*. I am sure that, had King Solomon said that, he could not have said a wiser thing." But the statesmen of his time, the followers of that *haute politique* which philosophised about the balance of power, and formed petty cabals in the drawing-rooms of the "Leo Hunters" of the day, contemptuously stigmatised his policy as "a policy of sewage." Over twenty years roll by, and the other day the Marquis of Salisbury went down to Oxford to plead for the Radcliffe Infirmary. He said : "I believe that if you respond munificently to the appeal that is made to you you will do something more than place this infirmary in a position of which it need not be ashamed ; you will be taking a long step towards introducing more closely the cultivation of one of the greatest of sciences—the science of medicine—in this ancient university. I always think that science has scarcely received amongst us all the tribute that it ought to receive amongst the sciences which rest upon observation. It is the most sober, the most absolute, the most positive amongst all the sciences. Again, there is no other science which is but another name for a work of mercy ; there is no other science that is so closely linked with the relief of human suffering as a remedy for human calamity in its most overwhelming form. It is a curious coincidence, a curious fact, that at this moment the whole tendency of scientific thought appears to be rapidly concentrating itself upon the fields in which medicine reigns supreme. In the early part of this century the infinitely small, or mainly the inanimate portion of creation—the atoms and all that surrounded them—ran supreme in the minds of scientific men. In the middle of the century it was something more delicate still—the discovery of the spectro-scope, and the meaning and interpretation of the infinitely small waves of that light whose nature we are not yet able to discern. Now, partly under the pressure of human necessity, there is another portion of the infinitely small—the bacilli—which is attracting more and more the attention of the scientific intellect of



Europe. It is dangerous always to prophesy, but I do not think anyone who has watched the course of science will doubt that for a generation to come the investigation of these creatures, which have been revealed to us by new methods of research and by singularly patient labour, and upon which the lives of millions of human beings depend, will figure larger in the scientific field than any other object of study, and these are the special domain and privilege of medicine."

Gentlemen, the man who spoke these words followed in his early life the profession of letters. In his own magnificent home his relaxation is chemistry. He is the head of one of the noblest families of this land, and until recently he was Prime Minister of State. No man in so great a position has ever spoken of our profession in terms of such admiration and respect. To the sailor accustomed to watch with intensest eagerness the minutest changes of sea and sky it will often happen that the appearance of the tiniest ripple, the look of a cloud no bigger than a man's hand, are things of profound importance. I regard this speech of Lord Salisbury as full of meaning. It is a recognition in high places of the value of our art to our country at large. We are accustomed at hospital dinners and dispensary bazaars to hear from great noblemen—yea, even from Princes of the Blood—what good fellows we practitioners are. *Mais ça ne fait rien*. It is kindly meant and we take it kindly. But the speech of Lord Salisbury refers not to our worth as individuals, but to the public value of that profession of which even the humblest private in its ranks is proud: yes, gentlemen, and whose honour he has often died to maintain. I look forward with confidence to the time when important public duties will fall to the lot of members of our profession, and when many posts of honour and distinction will be filled by them. Even at this moment is not a medical man holding a high and prominent position in the Government of the country—Sir Walter Foster? But, looking forward, as we all must do, to a greater position for medicine in the future than ever she has had before, will it not be incumbent on her followers, more than ever, to show themselves worthy of her? And the thesis, which I am here to maintain to-night, is that we shall not be worthy of her unless we are something more than mere prescribers of physic and healers of wounds. In my youth I had it strongly recommended to me to stick to my profession and leave everything else



severely alone. The life of a medical man was to see patients, do operations, order drugs, and collect fees. I thank God that I entirely repudiated this idea of my profession. If I had adopted it I should never have had the honour of being asked here to-night. I hold that an excessive devotion to the literature and work of our profession is a bad thing for us. It narrows us down. Life becomes made up of "cases." I am quite certain, from my own recollection and experience, that a teacher who knows nothing but the details of that section of the healing art which he practises never has the influence over his students that a man of general culture and catholic reading possesses. The latter has more humanity in him. He takes a broader view of affairs. He has learnt that there are other things in heaven and earth than such as can be heard through a stethoscope, or seen through a microscope, or laid bare by a scalpel. Therefore I say that this knowledge gives him power over the minds of his students, who irresistibly feel that he is not merely a great healer, that he is not merely an able teacher, that he is something more: that he is a widely educated man. What shall I say of those who do the general work of our calling? Some there are literally earning their bread by the sweat of their brow amongst the poor of our overgrown cities; some there are toiling in the blackness and darkness of mining or manufacturing villages; some there are braving the storm, the rain, the frost, on the bleak roads of a wild country practice. To these men, if they would but learn to love reading, a solitary shelf of well-picked books would alone be enough to lighten and sweeten their lives.

Some fourteen years ago a very clear-headed, common-sense, worldly-wise doctor, Milner Fothergill, wrote some very hard things about the social status of our profession, which he said was not equal to that of others. He unfortunately gave utterance to some remarks which were scarcely justifiable in the phraseology in which he put them. And so there fell upon him a terrible host of gentlemen, who wrote furious small-print letters in the medical journals denouncing him as a traitor to his colours—a bird fouling his own nest. On account whereof Fothergill for a while was very unpopular with that variety of practitioner who is always so exquisitely sensitive about the dignity of his profession. But I remember very well that the impression left upon me was that the furious letter-writers quite lost sight of the general bearing of

Fothergill's just and courageous remarks and lashed themselves into a fiery indignation solely on account of a few indiscreet sentences which he had suffered to break loose. Now I maintain that he was in the main right. To become a gigantic mutual admiration body is a mistake. There can be nothing worse for us than to be ignorant of our weak places, and the man who, like Fothergill, points them out to us is certain to be a thousand times more keenly alive to the real dignity of our profession than the vulgar persons who boast so much about it and add so little to it. By mere virtue of our profession we do not rank socially with other professions. The most callow curate with his Oxford B.A., the youngest sub-lieutenant of a marching regiment or a gunboat who wears Her Majesty's uniform—"Mr. Junior" of the Bar mess—by mere virtue of his cloth is taken into any drawing-room in the land. It cannot be said that this is the case with the medical man. His profession alone will not take him anywhere. He has to make his social position for himself. That he can do so, and that he can hold his own with anyone is apparent everywhere. Nay, I will go further, and say that no man commands more social respect than a well-bred, well-read practitioner. His education covers a great range of subjects and embraces more than that of other professions. It makes him a more entertaining and companionable friend, a more valued and respected guest. Even the querulous Pope said of us: "They are in general the most amiable companions and the best friends as well as the most learned men I know." So much the more reason, then, why our whole profession, down to the youngest graduate, should be men of such good general culture that their company should be welcomed not merely by the rich (for of these I make little account), but by all those whose well-trained minds, whose liberal ideas, and whose refined manners constitute them the true society of our country.

The other day I went to pay a visit to one of my oldest and kindest medical friends. For half a century he laboured in general practice, and retired some seven or eight years ago with a modest but sufficient competence. I found him, at 80, not strong, but upright in carriage, with hearing perfect, and an eye that can read the smallest print. When I came in upon him in the afternoon, two or three old friends were taking tea with him, and merry grandchildren were bringing him his cup and his plate. I went with him after a while to have a chat and a cigar in his study. I

knew him always as a reader, and so I was not surprised to find "Joseph Andrews" lying on the table, nor to hear him say that he was just going through Fielding again, and that the oftener he read him the more he enjoyed him. We talked about friends and old times and practice, and many things besides, but we got back eventually to reading, and his last words were: "Not for a thousand a year would I lose my love of reading."

Gentlemen, when we come to 80, may the evening of our days, like that of my old friend's, be made supremely happy by troops of friends and the love of good books.



## CLINICAL EVENINGS.

---

*November 14th, 1892.*

### A CASE OF TUBERCULAR ULCERATION OF THE PHARYNX SUCCESSFULLY TREATED WITH LACTIC ACID.

By PERCY KIDD, M.D.

ON the posterior wall of the pharynx there is a large and somewhat puckered, white cicatrix with some surrounding vascular injection, and a small nodule is seen on the right side. The left posterior pillar of the fauces is thickened and adherent to the posterior wall. Its surface presents a pale, greyish-pink, granular appearance. The corresponding pillar on the right side is in the same condition, but is not adherent to the back of the pharynx.

Both tonsils show a ragged pale-pink surface. At the base of the tongue on the right side there is a small, nodular swelling which is slightly ulcerated at its centre.

The larynx presents the following conditions: Epiglottis swollen, slightly reddened, and the free edge ulcerated; aryepiglottic folds and arytenoid regions of pale pink colour, and somewhat swollen; vocal cords normal.

There are signs of infiltration of the apex of the left lung, and the sputum contains tubercle bacilli in small numbers.

*History.*—Annie C——, aged 41, married, seven children. Cough, expectoration, pains in the chest, and wasting commenced about Christmas, 1891. In the following spring she became an out-patient under Dr. Acland, at the Brompton Hospital, when signs of phthisis were detected at the left apex. Throat symptoms, which had been present for two or three months, now became very severe, and the patient was sent to me at the Throat Department of the hospital in May last. The patient was then extremely wasted, anæmic, and feeble, and complained of agonising pain in the throat on swallowing, which almost entirely prevented her from taking food.

The pharynx was then extensively ulcerated on its posterior and lateral walls, the surface of the ulceration coarsely granular, marked with bright red points, and coated with thick greyish-yellow secretion. The tip of the epiglottis was slightly swollen and congested, but the larynx in other respects was healthy.

After thorough cocainisation, lactic acid (50 per cent. solution) was rubbed into the ulcerated surface, at first once a week, afterwards twice a week. The pain on swallowing was somewhat mitigated after a few applications, but it was not till the fourteenth application that the ulceration showed definite evidence of healing and cicatrisation. The concentrated pure lactic acid was now substituted, and slow but progressive improvement ensued. Twenty-two applications of lactic acid have now been made, extending over a period of five months.

Unfortunately, while the pharyngeal disease was mending, the epiglottis became more swollen, and ulceration has recently developed. Lactic acid has been lately applied, and some improvement is already noticeable.

The general treatment has consisted in the use of cod-liver oil and an alkaline bitter tonic. The pain in the throat has been relieved by lozenges of cocaine (gr.  $\frac{1}{8}$ ), and by brushing with menthol (20 per cent. solution in olive oil), before meals.

## TWO CASES OF LEFT INGUINAL COLOTOMY.

By D. H. GOODSALL, F.R.C.S.

MR. GOODSALL exhibited a clerk, aged 47, on whom he operated in January, 1890, for fissure of the anus, and he then found a tumour growing from the sacrum and extending into the pelvis on the left side. At the end of October, 1890, the abdomen began to show signs of distension, and on November 10th, 1890, a left inguinal colotomy was performed. Two days later the bowel was opened by a linear incision, and a month afterwards he left the hospital. He had since increased much in weight, and at the present time the characters of the tumour appeared to be unchanged.

MR. GOODSALL likewise showed a French polisher, aged 70, who had been the subject of cancer of the rectum. He had lost flesh

for two years, and had passed blood from the rectum for about three months, there being about six motions daily, accompanied by prolapse. The carcinomatous growth occupied the posterior three-fifths of the rectal wall. It began about 1 inch above the anus, and extended upwards for 3 inches, being freely movable. Left inguinal colotomy was performed on June 14th, 1892, and a longitudinal incision was made into the bowel two days later. On June 28th, 1892, the lower end of the rectum was removed together with the growth, a small incision being made behind the anus, a pair of blunt-pointed scissors only were thus used to separate the rectum from the surrounding tissues, section of the gut being performed by a wire *écraseur*. The patient left the hospital thirty-six days after the removal of the growth. The site of the anus was occupied by a scar, and the man had since gained in weight.

## “CAISSON” WORK IN BLADDER SURGERY.

By E. HURRY FENWICK, F.R.C.S.

MR. HURRY FENWICK gave a description of what he called “caisson” work in bladder surgery. To remove a small growth, he made a limited anterior incision into the bladder, and then sunk in an open cylinder somewhat like a Fergusson’s speculum; this being pressed against the floor of the bladder over the growth, the water was sucked out, and he operated under the direct influence of a powerful electric light. For these sessile growths under the old method a large incision into the bladder was required, together with a big rectal bag. With the method he demonstrated it was not only possible to remove the growth, but also to apply the cautery with exactitude. He exhibited two patients from whom he had removed sessile villous growths by this method.

## CASE OF RADICAL CURE OF HERNIA.

By C. B. LOCKWOOD, F.R.C.S.

MR. LOCKWOOD showed a case to exemplify some points in operations for the radical cure of hernia. The patient was 40 years old,



and had genu valgum. On the right side he had a very large scrotal hernia, and on the left a smaller one; he also had a small umbilical hernia. The scrotal herniæ were of ten years' duration, and prevented him from following his occupation, which is that of a toy maker. The largest and strongest trusses made by the City of London Truss Society failed to keep the ruptures up. The right hernia was operated upon first. The sac contained the cæcum, vermiform appendix, and the end of the ileum; its mouth was very big, and was secured with a sponge thrust into the abdomen. A quantity of large veins and the constituents of the cord were adherent to the back of the sac, and had to be torn off. The first point exemplified was that an operation of this kind could all be done through a small incision coterminous with the inguinal canal, but that stripping off the veins caused phlebitis, and subsequent orchitis. On the left side a similar operation was done, but the cord was free, and no orchitis resulted. Next, the simplicity and safety of these operations was exemplified. Both wounds healed under a single dressing, and, except for the orchitis, the patient experienced no discomfort. The effects of neglect were also exemplified. On the left side, where the inguinal canal had not been obliterated the operation was easy, and the operation gave a very strong occlusion; on the right side there was no inguinal canal, and the operation was difficult and less certain. Further, as the general condition of the patient was so defective, with probable prolapse of his mesentery, peritoneum, and kidneys (it was thought that the kidneys could be felt), the patient was advised that the operation was not a curative one, and that he would be well advised to wear a light truss when engaged in active operations.

## A CASE OF BONY TUMOUR OF THE ASCENDING RAMUS OF THE ISCHIUM ASSOCIATED WITH DISEASE (CHARCOT'S) OF THE HIP.

By GEORGE R. TURNER, F.R.C.S.

THE patient, J. B——, a sailor, aged 38, first noticed at sea in December, 1891, that his right knee was swollen. This was followed by swelling and pain in the right hip and thigh. He was obliged to give up work for the rest of the voyage, and could

only get about on crutches. He came to the Seamen's Hospital in April, 1892, with a history of gradual shortening of his right leg. There was no history of syphilis or rheumatism. He admitted to having had gonorrhœa; never any injury. He presented on admission no symptoms of tabes. There was eversion and shortening of the right lower extremity: the top of the trochanter major touched Nélaton's line. Muscles of the right thigh wasted and flabby. The knee was quite normal. He had been in the hospital some four weeks before some thickening and irregularity about the rami of pubes and ischium was noticed. This increased somewhat rapidly so that in time a well-marked bony tumour connected with the ischium could be felt. There were still no symptoms of tabes. He had no difficulty with micturition or defæcation. Mr. Turner, on June 28th, 1892, removed a bony tumour the size of a small Tangerine orange, which was growing from the ascending ramus of the ischium. There was another bony mass nearly loose in the cellular tissue separating the adductor muscles. The wound healed by first intention. The eversion of the limb could now be rectified, and the patient, who previously had to use crutches, was again able to walk. In September, rapid effusion into the hip joint occurred with well-marked crackling on movement. The shortening of the limb progressively increased to as much as  $3\frac{1}{2}$  inches. This was its extent when the man was shown to the Medical Society. On pulling on the limb it could be entirely rectified, and was obviously due to displacement of the altered head of the femur on to the dorsum ilii. There was free crackling on moving the hip, but no pain. The pupils responded to light, but there was entire loss of the patellar reflex, and some slight unsteadiness on standing with the eyes shut. There never had been any gastric crises or lightning pains in the limbs. There was no loss or alteration of sensation. His micturition, except for some delay at its commencement, was normal. Mr. Turner thought the chief interest of the case consisted in the association of the bony tumour with the disease of the hip. The latter, although the evidence of tabes was not complete, was probably Charcot's joint disease. The bony pelvic tumour growing from the ascending ramus of the ischium was some distance from the joint. Was it a mere coincidence, or did such bony enlargement accompany such cases of joint disease?

## CASE OF LEPROSY IMPROVING UNDER TREATMENT.

By P. S. ABRAHAM, M.D.

DR. ABRAHAM re-exhibited a lad whom he had shown last year suffering from leprosy, which had improved under treatment. The diagnosis had been confirmed on his arrival from Demerara, and since then there had been a marked retrogression in the manifestations of the disease. He had been at first submitted to injections of tuberculin, had regularly taken chaulmoogra oil, plasters of chrysarobin had been applied locally, and at present he was taking cod-liver oil together with 16 grains daily of gyno-cardate of magnesia. His present diet included fish.

The PRESIDENT referred to several cases under his care which he had treated on a simple plan with nux vomica, small doses of arsenic, together with a plentiful nutritious diet, and all had improved. He had very little faith in the drugs used, and believed the more important factors to be the climate and the diet. All the cases he believed to be absolutely non-contagious.

## CASE OF PULSATILE TUMOUR OF THE NECK.

By G. A. HERSCHELL, M.D.

DR. HERSCHELL showed a woman, aged 70, with a pulsatile tumour of the neck, which she had noticed for three years. He believed it to be a fusiform dilatation of the carotid, and the only symptoms attributable to it were occasional fainting fits.

[*N.B.*—The patient subsequently developed a similar swelling on the opposite side, and two months afterwards suddenly dropped down dead. No *post-mortem* could be obtained.]

## CYSTIC TUMOUR OF THE AURICLE.

By A. MARMADUKE SHEILD, F.R.C.S.

MR. SHEILD showed a cystic tumour of the auricle in a man, aged 44, which had existed since 1864. He believed it to be of sebaceous origin.



## ULCERATION OF THE EDGES OF THE EARS.

By A. MARMADUKE SHEILD, F.R.C.S.

MR. SHEILD likewise showed a youth, aged 17, the subject of ulceration at the edges of the ears, which had come on in the summer. There were no signs of Raynaud's disease.

---

*January 30th, 1893.*

## ILLUSTRATIONS OF VARIOUS DISEASES.

By the President, JONATHAN HUTCHINSON, F.R.C.S., F.R.S.

THE PRESIDENT exhibited some recently executed drawings and photographs illustrating various diseases. He mentioned that there was now a photographic department at the Royal College of Surgeons where any case of interest could be photographed free of charge, and where duplicates of portraits taken there could be obtained for lectures or other purposes. He showed a specimen of fracture of the fibula accompanied by complete displacement of the foot outwards, which was kindly lent by Mr. Thomson, of Dublin. He also exhibited a photograph of a man suffering from gout, showing a large deposit of uric acid in the iris, unaccompanied by iritis. The patient's hands were deformed with tophi. He exhibited the painting of a case of cancer following on eczema of the face, the latter being a sun eruption in a patient from the West Indies. Another drawing showed indurated scrofulous tumours on the legs of a girl, the affection described by Bazin as erythema induratum. Another series of water-colour drawings illustrated the condition known as urticaria pigmentosa, which began, he believed, in flea-bites, and which usually vanished in adult life. Another illustration represented herpes zoster extending down the leg quite to the ankle. Then there were illustrations of multiple cartilaginous tumours connected with the digits; of a condition of granuloma of face following inoculation from "greasy

hoof" in a horsekeeper; two photographs of congenital absence of the tibia and of the ulna; a picture of multiple malignant tumours of the scalp occurring in association with ordinary sebaceous cysts; and a photograph of an aneurysm on the side of the face.

## UMBILICAL FISTULA IN AN INFANT.

By LEONARD G. GUTHRIE, M.B.

DR. GUTHRIE showed the intestines of an infant, who died at the age of 6 weeks from the effects of umbilical fistula. The latter was a pervious Meckel's diverticulum leading from the ileum 12 inches above the ileo-cæcal valve, and opening on the surface of the abdomen. He thought that the specimen threw some light on the method of development of umbilical polypus.

## CASE OF EXTREME DEFECT OF SPEECH IN A BOY.

By WALTER B. HADDEN, M.D.

DR. HADDEN showed a boy, aged 6, who had an extreme defect of speech. He was intelligent above the average, had no local defect in the mouth, and was not deaf. He could pronounce the labials and dentals, but not gutturals, and sounds from the back of the throat he found unpronounceable. The case was quite curable by education, and was similar to one he had shown at the Neurological Society.

Dr. HALE WHITE said the case was similar to those shown by Mr. Golding Bird and himself at the Royal Medical and Chirurgical Society. They had preserved phonographic cylinders of those cases. The patient shown did not, like those cases, so constantly reproduce the same sound for the same word. Dr. Perry had suggested the name of "idioglossia" for the disease. They had been taught to speak by the aural method. It was interesting to note that though they had their own language yet they wrote to dictation correctly.

Dr. HADDEN, in response to the President, said that he had seen altogether some six or seven cases, each being the only one of the family affected.

## CASE OF COMPOUND DEPRESSED FRACTURE OF THE SKULL.

By HERBERT W. ALLINGHAM, F.R.C.S.

MR. HERBERT ALLINGHAM showed a lad who had been brought to the Great Northern Hospital with a compound depressed fracture of the skull. There was considerable hæmorrhage from the middle meningeal artery, with laceration of the brain. After picking out the comminuted fragments, a large aperture was left, which he covered over with a flap of periosteum turned down from the healthy bone above. The boy made an excellent recovery.

## TWO CASES OF INGUINAL COLOTOMY.

By HERBERT W. ALLINGHAM, F.R.C.S.

MR. HERBERT ALLINGHAM likewise exhibited two cases, both females, aged 33 and 42 respectively, admitted with stricture and ulceration of the rectum. He performed inguinal colotomy, and cut off a very large length of sigmoid colon from each, in order to prevent prolapse, with, in each case, a satisfactory result.

## CASE OF KELOID.

By CHARLES STONHAM, F.R.C.S.

MR. STONHAM showed a boy, aged 7, who two years ago sustained a severe burn; four months afterwards, keloid formation commenced in the scars. One of the patches was slowly disappearing, but the others remained stationary.

## CASE OF CONGENITAL SACRAL TUMOUR.

By CHARLES STONHAM, F.R.C.S.

MR. STONHAM likewise exhibited the sister of the above boy, aged 4 months, with a tumour, mainly over the left buttock, but reaching to the middle line. It was hemispherical in shape, and



extended down to the folds of the nates. It was soft to the touch, was attached to the deeper parts, and was painless on manipulation. At the lower segment was a small, nipple-like projection. He thought it was probably a fibro-cellular tumour with a considerable quantity of fat.

## A CASE OF ULCERATION OF AURICLES.

By C. B. LOCKWOOD, F.R.C.S.

MR. LOCKWOOD showed a case of gangrene of both ears, in which the patches of gangrene were symmetrical and similar to those described by Raynaud. At a first glance the patches might be mistaken for the results of frost bite, of this there was, however, no history. The disease began gradually during the mild weather, and though there was no anæsthesia, yet the pupils were unequal and did not react well to light, suggesting a trophic nerve lesion. There was absence of albumen and blood pigment from the urine. He concluded with some remarks in reference to the treatment.

## CASE OF ACNE-KELOID (BAZIN) [DERMATITIS CAPILLARIS (HEBRA)].

By JAMES STARTIN.

THIS case, a young man, aged 27, came under my observation at the London Skin Hospital, Fitzroy Square, on the 29th of November, 1887, telling me the disease began as an attack of acne in 1884. He had been under various treatments until then. He was then under my care for a few months, under the ordinary treatment for acne. I then lost sight of him until a month ago, and found him in the condition in which you have now seen him. In the 'Pathological Society's Transactions' of 1882, Mr. Marrant Baker's case with illustration more nearly approaches the character of the numerous keloids in this case; and in the same Society's 'Transactions' of 1883, Mr. Clutton describes a case following lupus and subsequent development of keloid in other operation wounds; and in 1884 Mr. Roger Williams quotes a case. The cases quoted by Professor Kaposi in his chapter on Framboesia I do not recognise as identical with these cases of true acne keloid

—a conjoint disease having all the main characteristics of both (Hutchinson). The disease seems to me to be “a chronic inflammation of the corium on the site of the acne induration, in consequence of which a free growth of connective tissue and blood vessels with papillary outgrowths in keloid form result.” Why they take on this keloid growth I cannot say. On manipulation the tumours are well defined, firm, and elastic, and on microscopic examination show in the deep part of the corium a dense fibrous network with long oval nucleated cells, permeated by numerous large vessels, but there is no sign of a small cell infiltration in the mass, and the epidermis in both superficial and deep layers is normal.

The reason I brought this case before the Fellows is that it is unique in the multitude and size of its keloids.

## CASE FOR DIAGNOSIS.

By A. MARMADUKE SHEILD, F.R.C.S.

MR. SHEILD showed a case for diagnosis. It was that of a youth, aged 16, who had been subject to rheumatic pains. The right hand and arm were getting weak, and the fingers were cold and glossy; the pulse was the same on both sides, but the neck was held very stiffly. In the right subclavian triangle was a distinct bony formation. In discussing the diagnosis he excluded the idea of a cervical rib; considered the possibilities of its being myositis ossificans, or diffuse osteoma, springing from the vertebræ, but inclined to the idea that it was probably a case of “quiet” cervical caries, with hyperostosis round the diseased parts.

## CASE OF PSEUDO-HYPERTROPHIC PARALYSIS.

By W. PASTEUR, M.D.

DR. PASTEUR showed a boy, aged 17 years, the subject of pseudo-hypertrophic paralysis. The case was chiefly remarkable in that the disease appeared to have become arrested.

H. R——, aged 17. Had always been delicate. His father was an epileptic, and his mother weakly. He has five brothers alive, who are all delicate and anæmic. Of two sisters living, one is

consumptive, the other anæmic. In childhood, H. R—— was unable to play about as other children did. He never could run, and had a little trouble in rising from the sitting posture. He began to walk at eighteen months. When he was 12 years old, he obtained a situation at a stationer's, involving light work and little moving about, and kept it for several years. He recently gave it up, on account of fatigue from the constant standing it involved. His mother states that he has always had very large calves, and very thin thighs.

*Present Condition.*—There is typical hypertrophy of both calves, the effect of which is heightened by contrast with the very wasted thighs. The infra-spinati are also distinctly enlarged and hard. With the exception of the extensors of the forearm, the upper limb muscles are generally flabby and wasted. The gait is not very characteristic, and it is an interesting fact that he has attended the out-patient departments of several general hospitals at various times during the last seven years, apparently without arousing any suspicion of the true nature of his disease. There is well-marked lordosis, and he exhibits in a slight degree the characteristic manner of rising from the recumbent posture. The hypertrophied muscles react to moderate faradic and galvanic currents, but the thigh muscles contract only to strong currents.

---

*February 27th, 1893.*

## CASE OF LEPROSY IN COURSE OF RECOVERY.

By the President, JONATHAN HUTCHINSON, F.R.C.S., F.R.S.

THE PRESIDENT exhibited a patient who was recovering from leprosy, and who had been under his observation for two or three years. He had lived abroad, and had become the subject of ordinary leprotic patches on the hands and feet, which had extended upwards. The ulnar nerves were so enlarged as to be easily felt. At the present time all the patches had disappeared, but the hands and feet were insensitive to heat and cold, and also to pain. The treatment had been long continued, small doses of arsenic, and the



entire absence of fish from the diet. He had under observation another similar case, which had also recovered as far as recovery was possible. Recovery was the rule in mild cases treated in the English climate with regulated diet. He held that a diet of fish was of primary importance in the production of the disease, and that, though English fish was not able to start the disease *de novo* in a healthy person, yet a fish diet contributed to its development.

## ACUTE OSTEO-MYELITIS OF EACH HUMERAL DIAPHYSIS; DOUBLE RESECTION; RECOVERY.

By EDMUND OWEN, F.R.C.S.

MR. EDMUND OWEN showed an infant which had suffered from double acute septic osteo-myelitis of the humeri at their upper ends. He stated that on June 10th, 1892, a male child, 2 years 1 month old, was brought into the Hospital for Sick Children, in a state of extreme exhaustion. The mother said that a little more than a fortnight previously he had had a series of fits; that since then he had been feverish and unable to eat, and that on the day before his admission she had noticed that the shoulders were swollen, hot, and painful. On admission, the child was found to be somewhat rachitic, and very anæmic and ill. The temperature was 104·4°. The skin over each shoulder was tense, red, shining, and hot, and marked with distended veins. Fluctuation could be obtained across the joints. The case was diagnosed as one of acute septic inflammation of the upper end of each humeral diaphysis, with implication of the shoulder-joints. The child was therefore at once taken into the theatre, and when he was anæsthetised Mr. Owen opened one abscess by cutting through the deltoid, whilst the surgical registrar, Mr. Wagstaff, did the same for the other. On both sides the joints contained pus, the cartilage of the humerus being extensively destroyed. The head of each humerus was resected, and the end of the diaphysis was scraped out. The joints were then washed out with a solution of chloride of zinc, drainage-tubes being inserted. The wounds were dressed with wood-wool, and the arms were fixed to the sides. The child made an uninterrupted recovery, the joints being left freely movable.

Mr. Owen remarked that acute septic osteo-myelitis is one of the most serious conditions which can be met with in children. The inflammation is usually preceded by an injury which has lowered the vitality of the bone, or by some fever which has left the child in an enfeebled condition, so that the micrococci (which are probably lurking in or about every child) are enabled to obtain a foothold in, and spread devastation through, the tissue. The delicate new bone at the end of a diaphysis is the favourite seat of the disease, and in very little children the neighbouring joint may be quickly involved. So often, indeed, does this happen, that these cases were once grouped together to form, as it were, a special disease, under the name of "acute arthritis of infants." Subsequent experience has shown, however, that there is no need for such a classification. The lesion is not primarily a joint disease, nor is it an epiphysitis. It is at first a *diaphysitis* close to the junction-cartilage. Perhaps the best name for it is para-epiphysitis. Not a few of the subjects of the disease die outright from shock, whilst others succumb to acute blood-poisoning. If recovery does take place, it is usually at the expense of the joint, if not of the limb. The simultaneous affection of the two shoulders, as recorded above, points, I think, to the constitutional origin of the trouble (as opposed to that of local injury); whilst the recovery of an apparently moribund infant after resection of the upper extremity of each humerus shows once more the importance of prompt and vigorous treatment in every case, however apparently hopeless.

### CASE OF NON-MALIGNANT STRICTURE OF THE RECTUM, WITH PLEURITIC EFFUSION; THIRTEEN TAPPINGS; SUBSEQUENT LEFT ILIAC COLOTOMY.

By F. DE HAVILLAND HALL, M.D., and D. H. GOODSALL, F.R.C.S.

P. F——, female, aged 42, shoemaker, married, no children. In December, 1883, was operated on at St. Mark's Hospital, by Mr. William Allingham, for piles. In January, 1884, while at a convalescent hospital she had a fall which caused an ischio-rectal abscess and subsequently a fistula; in the following May, Mr. W. Allingham operated on her for fistula, and she was under his care in November, 1886, for stricture. In 1888, Mr. W. Alling-

ham, having resigned his surgeoncy at St. Mark's, the patient came under Mr. Goodsall's care. The patient first showed signs of syphilis in March, 1890; there was a rash on the face and arms; occasionally large quantities of blood from the rectum, from which there was a constant discharge, were passed. She was re-admitted into the hospital on the 11th October, 1890, when the stricture was dilated and a vulcanite tube introduced, the patient being discharged on the 3rd November, 1890. On February 27th, 1892, she was again admitted for colotomy, but her condition was so bad that the operation was postponed till her health had been improved. On April 3rd, 1892, Dr. de Havilland Hall, having been asked to see her because of gradually increasing dyspnoea, removed 57 ozs. of flaky serous fluid from the left pleural cavity (maximum temperature during the preceding fortnight,  $99^{\circ}$ ; minimum,  $97.4^{\circ}$ ) and from this date to the 12th July the patient was tapped thirteen times, 705 ozs. in all being removed from her left pleural cavity. On July 5th, left iliac colotomy was performed, a large quantity of ascitic fluid escaping during the operation; two days afterwards the colon was opened, and on the third day the bowels acted for the first time through the artificial anus. On the 5th October, the patient was discharged, the rectum being almost closed by the contracted stricture.

## CASE OF TRAUMATIC RUPTURE OF THE URETHRA.

By A. PEARCE GOULD, M.S.

MR. PEARCE GOULD showed a case of traumatic rupture of the urethra in a man, aged 41, who slipped from a ladder and fell astride an iron railing. On admission into the Middlesex Hospital the scrotum was distended and oedematous. A median incision was made, and the corpus spongiosum was found to be completely divided. A catheter was passed into the bladder, and the divided ends of the urethra were sutured together with silk. After the first night not a drop of urine escaped from the wound, the deep part of which healed by first intention. He referred to two other cases. The first case was that of a boy aged 7, in whom he sutured a completely divided urethra, and then closed the perineal wound, draining above the pubes; in this case the perineal wound healed at once. The second case was that of a man aged 27, who had



bladder trouble which led to a digital exploration through a perineal wound. The wound was not sutured, but a firm pad was applied; nevertheless not a drop of urine escaped through it, and it healed by first intention. The cases illustrated the advisability of stitching together a ruptured urethra and showed that it was not necessary to leave the wound open, one of them demonstrating that though the urethra might be ruptured completely across, yet the patient could pass urine as if nothing had happened.

## CASE OF CHRONIC ENLARGEMENT OF THE SPLEEN.

By EDMUND CAUTLEY, M.B.

DR. E. CAUTLEY brought forward a young woman suffering from chronic enlargement of the spleen. She was 26 years of age and the increase in the size of the spleen had been noticed since she was 11. It now extended down to the iliac fossa. The red blood-corpuscles were only half their normal amount, and the proportion of white to red was as 1 to 100. There was no history of malaria, syphilis, or of hæmorrhages.

The main points raised in connection with the case were the questions of causation and prognosis.

## A PROBABLE SYPHILIDE REFRACTORY TO TREATMENT.

By P. S. ABRAHAM, M.D.

DR. P. ABRAHAM showed a patient, aged 58, with an eruption on the face, which had proved refractory to treatment. He had regarded it as probably syphilitic because of its rapid development, its serpiginous raised margin, the patient's history of numerous miscarriages, and the favourable action on it of iodides when first administered. It had recently altered much in character.

The PRESIDENT looked upon it as lupus rather than syphilis, because the latter was attended by more inflammation and less growth.

Dr. COLCOTT FOX regarded it as true lupus—a tuberculosis of the skin of the face.

Dr. HERON suggested the injection of tuberculin to make the differential diagnosis.

## CUTANEOUS NÆVUS OF LEFT UPPER EXTREMITY, WITH GENERAL ENLARGEMENT OF THE LIMB.

By MONTAGUE MURRAY, M.D.

E. W——, aged 21, labourer. Has had from birth a cutaneous nævus involving the whole of the left upper extremity. The upper half of the left side of the thorax is largely covered with the same kind of skin. The nævus in some places reaches, but does not cross, the mid-line. The hand and lower part of the forearm are generally blue, the rest bluish-red, but patient states that some years ago the whole extremity was of the bluish tint. The bones of the limb are larger and the muscles somewhat stronger than on the opposite side. The man is not left-handed, but he is convinced by experience that he can reach farther and accomplish harder work with his left than with his right arm.

	R.	L.
<i>Length</i> (acromion to finger tips).....	32·00 in.	33·5 in.
„ ( „ to olecranon) elbow flexed.	15·5 „	16·0 „
<i>Width</i> between condyles of humerus.....	3·1 „	3·35 „
„ „ styloid processes .....	2·4 „	2·5 „
Circumference of arm.....	10·75 „	11·0 „
„ wrists .....	7·2 „	7·6 „
Dynamometer (average of six) .....	80 lbs.	115 lbs.
Water displaced by hand and forearm .....	84 ozs.	100 ozs.
„ „ hands alone .....	18 „	23

## CASE OF DUPUYTREN'S FRACTURE.

By JONATHAN HUTCHINSON, Jun., F.R.C.S.

MR. J. HUTCHINSON, jun., showed a man with supposed Dupuytren's fracture. The accident happened 16 months ago, and there was much difficulty in the reduction. The limb had been constantly and increasingly painful since. There was an increase of an inch and a half in the distance between the tibia and fibula at their lower ends, with only slight movement at the ankle-joint.

## CONGENITAL TUMOUR IN AN INFANT.

By A. MARMADUKE SHEILD, F.R.C.S.

MR. SHEILD showed an infant, 6 months old, which had at birth a tumour in its pectoral region. The tumour did not appear to communicate with the pleura, was partly lobulated, and was, he believed, a fibro-fatty growth.



## CONGENITAL WARTY GROWTHS OF THE HAND AND FOOT.—FUNGATING SORE.

By A. MARMADUKE SHEILD, F.R.C.S.

MR. SHEILD showed two further cases: one of congenital warty growths of the hand and foot, and the second that of a large fungating sore about the anus in an elderly man, which was at first regarded as epithelioma, but which had rapidly disappeared under antisiphilitic treatment.



## CURED MENINGOCELE.

By STEPHEN PAGET, F.R.C.S.

MR. STEPHEN PAGET exhibited a man with a cured meningocele in the situation of the posterior fontanelle.

---

*April 17th, 1893.*

## EXCISION OF THE CLAVICLE FOR SARCOMA.

By W. F. HASLAM, F.R.C.S.

MR. HASLAM (Birmingham) showed a man, aged 31, whose clavicle he had completely excised five months ago for sarcoma. The patient had noticed, about nine weeks before his admission to the hospital, that lifting weights at his work caused pain in his right clavicle; this pain continued for about a month, and then, for the first time, he felt a lump about the size of a walnut towards the sternal end of the bone; this gradually increased in size, and the pain diminished. On admission, a tumour was found on the right clavicle, oval in shape, and situated just externally to the sternal end of the bone, measuring  $4\frac{1}{2}$  inches horizontally, and  $2\frac{1}{2}$  inches vertically. The surface was smooth and firm, though its consistence varied somewhat in places, and here and there distinct crepitation could be elicited on pressure. As far as could be ascertained, the growth was principally on the anterior aspect of the bone, though it extended upwards and downwards. The skin was freely movable over it, and there was no evidence of any pressure on the subjacent venous trunks. There was no history of syphilis. Removal was effected by means of an incision along the whole length of the bone, and a short vertical one carried upwards over the most prominent part of the growth. The attachments of the deltoid and trapezius were next cut through, the acromio-clavicular joint was opened, and the conoid and trapezoid ligaments were divided; this allowed the outer end of the bone to be raised, so that the subclavius could be dissected off.

After division of the sterno-mastoid and pectoralis major, the sterno-clavicular articulation was cut through, the rhomboid ligament divided, and the bone removed. No hæmorrhage of any importance was encountered until a small portion of the growth adherent to the deep cervical fascia was being dissected off, when a medium-sized vein was cut just as it was perforating the fascia. A drainage-tube was inserted, and the wound dressed with blue gauze. Healing was somewhat prolonged, owing to suppuration taking place, but by providing a free exit for pus no harm resulted, and the temperature only once reached  $100\cdot6^{\circ}$ . At the present time the cicatrix is sound, and there is singularly little deformity resulting from the operation, the point of the shoulder being nearly as high as the other, and not appreciably nearer the middle line. The movements of the arm, though not yet as free as normal, are considerable. On examination of the tumour, the amount of growth was found to be small in proportion to the new bone formation, and its origin was doubtless in the periosteum, on the upper and anterior surface of the bone. Dr. C. F. Marshall reported that the superficial portion consisted of a small round-celled sarcoma infiltrating the fibrous layers of the periosteum. In the deeper portions there were trabeculæ of cartilage showing stages in the formation of bone. This, however, tended to break down rather than form perfectly developed bone.

## SARCOMA OF THE CLAVICLE.

By F. BOWREMAN JESSETT, F.R.C.S.

MR. JESSETT showed a specimen of sarcoma of the clavicle which he removed from a girl, aged 16, four years ago, and the details of which he published in 'The Lancet,' of June 1st and 8th, 1889. Several small recurrent growths had since been removed, and the patient, who had had no recurrence, in any way, for the last two years, was at present quite well and able to use the limb quite freely.

---

*April 24th, 1893.*

# A CASE OF NASAL POLYPUS OF UNUSUAL FORM AND SIZE, WITH SPECIMENS AND DRAWING.

By W. SPENCER WATSON, F.R.C.S.

DR. HUNT, of Yeovil, sent Mr. J. S—— to see me on December 6th, 1892. He was a tall, well-built man, somewhat spare, and with a nervous, depressed manner. His age was 50 years; his occupation none; but he had been a solicitor's clerk, and had spent some years in Vancouver, whence he returned about six years ago. Both sides of his nose are much obstructed, being almost completely blocked on the right side, and only partly so on the left. His sense of smell is quite lost on the right side, but not on the left. The nose is well formed and symmetrical, with the exception that the right ala is somewhat flattened and motionless. He has noticed this flattening of the right ala and the symptoms of obstruction for about two years. A flattened, leaf-like, polypoid growth was visible in the right anterior naris; none was visible in the left. Posterior rhinoscopy completely failed, but the finger passed into the naso-pharynx detected polypi occupying both sides of that cavity and obstructing both choanæ, though it seemed as if the growths came entirely from the right nostril, and this afterwards proved to be the case.

On December 6th, 1892, cocaine was applied by Mr. Henry Davis, who assisted me, and after trying various forms of snare with very little success, bringing away only a moderate sized portion of the polypi, I succeeded in looping the ring of the ring-knife round the main bulk of the growth (the largest piece in the specimen jar, and represented in the illustration) and extracting it. The three pieces in the centre of preparation-jar were removed at this sitting. The remaining pieces were removed partly by the wire snare and partly by means of the sliding forceps, at various dates between December 6th and December 21st, when the patient went home to Somersetshire, and was directed to use a spray of hâzeline and vulcanite plugs. The nostrils were at that time quite free from obstruction, and he was immensely relieved. The improvement, both in his local condition and in his general



health, continued, and when last seen, on February 28th of the present year (1893), he was quite well, and no obstructing growth was visible in either of the nasal fossæ.



Opposite views of the same specimen, removed with others from the right nostril, from drawings by Mr. George Spencer Watson.

Some of the specimens exhibited differ very little from ordinary polypi, though they are considerably flattened, and some of them

are pieces of hypertrophied membrane from the turbinals, but the central large piece offers several peculiarities of form which are well represented by the pencil sketch, which was made on the same day on which it was removed, and before it had been altered in appearance by immersion in spirit. It will be observed that the "finger-like processes" which form the main bulk of the mass, are now much more tapering, and less bulky in every sense, than when they were first removed.

Having compared this one with many museum specimens, I have only found one that resembles it—a large polypus, presented by Sir Astley Cooper to the College of Surgeons Museum (No. 3940 of the Pathological Series)—but in that specimen the main bulk of the tumour consists of a large cystic growth. There is no history of the case in the Museum Catalogue. At first I was inclined to think that the growths must have originated in some papillomatous hypertrophy of the turbinated bodies, but their form is perhaps better explained as the result of pressure.

The *diagnosis* as to the seat of the polypi was obscure, as it always is when the nasal fossa is so full of obstructing growths, that no view of the deeper parts can be obtained. It was also obscure as to whether one or both fossæ were involved. There was great obstruction on the left side, as well as on the right, and though there were no growths visible anteriorly in the left fossa, it was not till after the removal of the principal mass occupying the naso-pharynx that it became evident that the obstruction to the left side was due to the overlapping of the left posterior naris by the polypoid mass whose origin and attachments were in the right cavity only.

In the *treatment*, the failure of the snare method was due to the very irritable state of the naso-pharynx, which rendered it impossible to manipulate the loop of wire by passing the finger into the fauces. The ring-knife being a rigid loop did not yield before the closely packed processes, and I fortunately succeeded in slipping it well over the bulk of the growth and detaching it at a point from which the greater number of the growths sprang and spread out radially. The specimen and drawings both show this peculiarity very well.

[*N.B.*—The specimens are mounted, and can be seen in the Royal College of Surgeons Museum, Pathological Series, No. 3943A.]



## CASE AFTER AMPUTATION OF THE HIP-JOINT.

By H. P. SYMONDS, F.R.C.S. Edin.

*February 8th, 1890.*—W. Watts, aged 8, was admitted into the Radcliffe Infirmary suffering from hip-joint disease; on account of the rapid progress of the disease excision of the hip-joint was performed by the anterior incision.

*April, 1890.*—The joint was found distended with pus, and the cartilage covering the acetabulum was eroded by the disease. The neck of the femur was sawn through close to the trochanter major.

*October 19th.*—Patient was discharged to attend as out-patient.

*December 10th.*—Patient was re-admitted, suffering from necrosis of trochanter and apparently of the pelvis, the disease progressing very rapidly, and the temperature assuming a hectic type.

*January 13th, 1891.*—It was decided, in order to save the patient's life, to amputate the limb by Furneaux Jordan's method.

The acetabulum was found to be necrosed, and part of the dead bone was removed. At the operation his weak condition and state of collapse were such as to necessitate transfusion, and pressure forceps were left on the vessels to avoid the loss of time in securing them by ligature.

After the immediate shock of the operation was over the patient rapidly regained strength, and February 8th was up and about on crutches. He was sent to the convalescent home, and has since that time enjoyed perfect health.

He has since been fitted with an artificial leg by Messrs. Lindsey, and the chief point worthy of notice about him at the present time is the fact that he can walk about at ease without any further support, being, in fact, able to walk more than 2 miles without any discomfort. This result has been effected by the perfect way in which the artificial limb is fitted to the pelvis, by its lightness, and by the way in which compensating movement and lateral adjustment have taken place in the lumbar region. The stump of bone reproduced from the periosteum left after the operation does not seem to have any share in this result.

Senn records two cases in which a stump was left as large as after amputation of the thigh. I also hear a case was reported from St. Thomas's Hospital, but I am unable to find it, and there is no record of a patient being able to walk well without stick or crutch.



## CASE OF ARTERIO-VENOUS ANEURYSM.

By CHARLES STONHAM, F.R.C.S.

MR. STONHAM exhibited a woman, aged 21, who, ten years ago, received a blow over the metacarpal bone of the thumb. An arterio-venous aneurysm had formed between the arteria dorsalis pollicis and the commencement of the radial vein. There was a marked thrill, which disappeared on slight pressure, and also a distinct and peculiar bruit. It had caused pain only during the last four months. He thought he could detect a distinct sac lying beneath the radial vein. For treatment he proposed to apply elastic pressure over the tumour, together with proximal pressure on the radial artery; if this failed, he would cut down over the swelling and ligature the artery, both proximally and distally.

## CHANCRE OF THE CHEEK—INFECTION THROUGH A BITE.

By JONATHAN HUTCHINSON, Jun., F.R.C.S.

MR. JONATHAN HUTCHINSON, jun., showed a man, aged 35, who had received a bite on the cheek in the course of a quarrel. A small chancre developed, which was but slightly elevated and secreted but little; there were a large submaxillary bubo and an early roseolar rash over the body. The person who inflicted the bite had been attending as an out-patient with secondary syphilis. The patient had proved resistant to mercurials administered by the mouth, but was doing fairly well under inunction.

## PLASTIC OPERATION FOR RODENT ULCER.

By JONATHAN HUTCHINSON, Jun., F.R.C.S.

MR. HUTCHINSON, jun., also showed a man, aged 68, who had been the subject of rodent ulcer affecting the inner half of the right lower eyelid and the adjacent parts of cheek and nose. This was dissected out, and the raw area covered with a flap of skin turned down from the forehead, which had firmly united, and the patient had no epiphora.



# INDEX.

---

	PAGE
Abortions, tubal, and tubal moles (J. B. Sutton) . . .	48
ABRAHAM (P. S.) case of leprosy . . . . .	348
—— ——— probable syphilide . . . . .	358
Address, opening, by the President (Jonathan Hutchinson) .	1
—— by the Fothergillian Medallist, 1893 (W. R. Gowers) .	300
Albuminuria, chronic, some clinical varieties of (C. H. Ralfe) . . . . .	212
ALLCHIN (W. H.) <i>remarks</i> . . . . .	115
ALLINGHAM (H.) piles: the importance of recognising the varieties as determining the selection of treatment .	73
—— case of fracture of the skull . . . . .	351
—— cases of inguinal colotomy . . . . .	351
ALTHAUS (J.) <i>remarks</i> . . . . .	62, 271
Aneurysm, aortic, hæmoptysis in (T. Gilbert Smith) .	324
—— arterio-venous, case of (C. Stonham) . . . . .	367
Aphasia, from a fall, case of (C. E. Beevor) . . . . .	272
Auscultation, intra-thoracic (B. W. Richardson) . . . . .	31
 BANKS (W. Mitchell) physic and letters—the Annual Oration	327
BARLOW (T.) <i>remarks</i> . . . . .	63
BATTLE (W. H.) two cases of abdominal section for tumours	244
—— <i>remarks</i> . . . . .	251
BEEVOR (C. E.) case of aphasia . . . . .	272
—— <i>remarks</i> . . . . .	277
BIDWELL (L. A.) cases of skin-grafting for lupus . . . . .	242
—— <i>remarks</i> . . . . .	79
Bladder surgery, “caisson” work in (E. H. Fenwick) .	345
BRAIDWOOD (P. M.) <i>remarks</i> . . . . .	299



	PAGE
BRISTOWE (J. S.) on syphilitic affections of the nervous system—the Lettsomian Lectures . . . . .	116
—— — (President) <i>remarks</i> . . . . .	262, 326
BROWNE (G. Buckston) suprapubic prostatectomy . . . . .	226
—— <i>remarks</i> . . . . .	237
BRUNTON (T. Lauder) <i>remarks</i> . . . . .	224
Bubo, ulcerating, of the groin, hæmorrhage from (A. M. Sheild) . . . . .	318
CARR (J. W.) <i>remarks</i> . . . . .	63, 277, 300
CAUTLEY (E.) case of chronic enlargement of the spleen . . . . .	358
Chancre of the cheek, case of (J. Hutchinson, Jun.) . . . . .	367
CHEYNE (W. Watson) case of skin-grafting for lupus . . . . .	241
Cholera, its epidemic progression and causation (J. B. Hamilton) . . . . .	80
CLARKE (W. Bruce) treatment of lupus of the face by free removal and skin-grafting . . . . .	238
—— — (cases) . . . . .	241
—— <i>remarks</i> . . . . .	236, 244
Clavicle, sarcoma of, case of (W. F. Haslam) . . . . .	361
—— — — (F. B. Jessett) . . . . .	362
COLLIER (W.) athletic exercises as a cause of disease of the heart and aorta . . . . .	64
—— <i>remarks</i> . . . . .	72
Colotomy, inguinal, two cases of (D. H. Goodsall) . . . . .	344
—— — — (H. W. Allingham) . . . . .	351
—— left iliac, case of (D. H. Goodsall) . . . . .	356
CRIPPS (W. Harrison) <i>remarks</i> . . . . .	20, 187
CROCKER (Radcliffe) <i>remarks</i> . . . . .	242
CULLINGWORTH (C. J.) <i>remarks</i> . . . . .	56, 251
Diagnosis, case for (A. M. Sheild) . . . . .	353
Diseases, various, illustrations of (J. Hutchinson, President) . . . . .	349
DORAN (Alban) the surgical treatment of cysts of the vulvo-vaginal or Cowper's glands . . . . .	38
—— <i>remarks</i> . . . . .	28
Ears, case of ulceration of the edges of the (A. M. Sheild) . . . . .	349
—— — (C. B. Lockwood) . . . . .	352

	PAGE
Educational requirements in their relation to the constitutional differences between boys and girls (F. Warner) .	263
EDWARDS (F. S.) <i>remarks</i> . . . . .	78, 236
FENWICK (E. H.) "caisson" work in bladder surgery .	345
Fever, traumatic, physiology of death from (J. D. Malcolm) .	188
Fistula, umbilical, in an infant (L. G. Guthrie) . . . . .	350
Fracture, prevention of shortening after (C. B. Keetley) .	181
— Dupuytren's, case of (J. Hutchinson, Jun.) . . . . .	359
GOODSALL (D. H.) two cases of left inguinal colotomy .	344
— and HALL (F. de H.) case of stricture of the rectum with pleuritic effusion . . . . .	356
GOULD (A. Pearce) case of traumatic rupture of the urethra	357
GOWERS (W. R., Fothergillian Medallist, 1893) address on neurology and therapeutics . . . . .	300
GRIFFITH (W. S. A.) <i>remarks</i> . . . . .	58
Growths, warty, of the hand and foot (A. M. Sheild) .	360
GUTHRIE (L. G.) case of umbilical fistula in an infant .	350
HADDEN (W. B.) congenital syphilis as a cause of nervous diseases in children . . . . .	59
— case of extreme defect of speech in a boy . . . . .	350
— <i>remarks</i> . . . . .	64, 350
HAIG (A.) <i>remarks</i> . . . . .	223
HALL (F. de H.) and GOODSALL (D. H.) case of stricture of the rectum with pleuritic effusion . . . . .	357
— <i>remarks</i> . . . . .	223, 277, 299
HAMILTON (J. B.) cholera: its epidemic progression and causation . . . . .	80
— <i>remarks</i> . . . . .	72, 100
HANDFIELD-JONES (M.) pyrexia following the anæmia due to hæmorrhage . . . . .	312
HARRISON (Reginald) <i>remarks</i> . . . . .	236
HART (Ernest) <i>remarks</i> . . . . .	98
HASLAM (W. F.) case of excision of the clavicle for sarcoma	361
HAWKINS (F.) croupous pneumonia in children . . . . .	277
— <i>remarks</i> . . . . .	18, 225, 300
Heart and aorta, athletic exercises as a cause of disease of the (W. Collier). . . . .	64

	PAGE
Heart, the irregular; a clinical study (A. E. Sansom) .	100
Hernia, case of radical cure of (C. B. Lockwood) .	345
HERSCHELL (G. A.) case of pulsatile tumour of the neck .	348
Hip-joint, case after amputation of the (H. B. Symonds) .	366
HORROCKS (P.) <i>remarks</i> . . . . .	56
Humeri, case of osteo-myelitis of the (E. Owen) .	355
HUTCHINSON (Jonathan, President) opening address on names, definitions, and classifications . . . . .	1
— illustrations of various diseases . . . . .	349
— case of leprosy in course of recovery . . . . .	354
— <i>remarks</i> . . . . .	72, 78, 96, 187, 212, 235
— (Jonathan, Jun.) case of Dupuytren's fracture .	359
— — — chancre of the cheek . . . . .	367
— — — plastic operation for rodent ulcer . . . . .	367
JESSETT (F. Bowreman) case of sarcoma of the clavicle .	362
— <i>remarks</i> . . . . .	19
KEETLEY (C. B.) on the prevention of shortening after fracture . . . . .	181
— <i>remarks</i> . . . . .	188
Keloid, case of (C. Stonham) . . . . .	351
— acne, case of (J. Startin) . . . . .	352
KERR (Norman) <i>remarks</i> . . . . .	225, 271
KIDD (Percy) case of tubercular ulceration of the pharynx .	343
LEES (D. B.) <i>remarks</i> . . . . .	62
Leprosy, case of (P. S. Abraham) . . . . .	348
— — (J. Hutchinson, President) . . . . .	354
Lettsomian Lectures. On syphilitic affections of the nervous system (J. S. Bristowe) . . . . .	116
LEWERS (A. H. N.) <i>remarks</i> . . . . .	57
LOCKWOOD (C. B.) case of radical cure of hernia . . . . .	345
— — ulceration of auricles . . . . .	352
— <i>remarks</i> . . . . .	244, 323
Lupus of the face, treatment of, by skin-grafting (W. Bruce Clarke) . . . . .	238
— — (cases of) . . . . .	241, 242



	PAGE
MACKENZIE (Stephen) <i>remarks</i> . . . . .	114, 222
MACLEOD (H. A.) <i>remarks</i> . . . . .	96
MACNAMARA (C. N.) <i>remarks</i> . . . . .	99
MALCOLM (J. D.) the physiology of death from traumatic fever . . . . .	188
—— <i>remarks</i> . . . . .	29, 212
MAUDE (A.) <i>remarks</i> . . . . .	113
Meningocele, cured, case of (S. Paget) . . . . .	361
Mental dissolution, symptoms of (G. H. Savage) . . . . .	252
MEREDITH (W. A.) the treatment of the peritoneum in ab- dominal surgery . . . . .	21
—— <i>remarks</i> . . . . .	30
Moles, tubal, and tubal abortions (J. B. Sutton) . . . . .	48
MORISON (A.) <i>remarks</i> . . . . .	299
MURRAY (M.) case of cutaneous nævus of left upper ex- tremity . . . . .	359
Nævus, cutaneous, case of (M. Murray) . . . . .	359
Neurology and therapeutics (W. R. Gowers) . . . . .	300
OPENSHAW (T. H.) <i>remarks</i> . . . . .	187
Oration, the, on physic and letters (W. Mitchell Banks) . . . . .	327
OWEN (Edmund) case of osteo-myelitis of the humeri . . . . .	355
PAGET (Stephen) case of cured meningocele . . . . .	361
Paralysis, pseudo-hypertrophic, case of (W. Pasteur) . . . . .	353
PASTEUR (W.) case of pseudo-hypertrophic paralysis . . . . .	353
—— <i>remarks</i> . . . . .	114, 262, 300
Peritoneum, treatment of the, in abdominal surgery (W. A. Meredith) . . . . .	21
Pharynx, case of ulceration of the (P. Kidd) . . . . .	343
Piles: varieties and treatment of (H. Allingham) . . . . .	73
Pneumonia, croupous, in children (F. Hawkins) . . . . .	277
Polypus, nasal, case of (W. S. Watson) . . . . .	363
PRESTON (T. J.) <i>remarks</i> . . . . .	72
Prostatectomy, suprapubic (G. Buckston Browne) . . . . .	226
Pyrexia following the anæmia due to hæmorrhage (M. Hand- field-Jones) . . . . .	312

	PAGE
RALFE (C. H.) on some clinical varieties of chronic albuminuria	212
—— <i>remarks</i>	226
RICHARDS (J. Peeke) <i>remarks</i>	262
RICHARDSON (B. W.) intra-thoracic auscultation: a new de-	
parture in physical diagnosis	31
—— <i>remarks</i>	113
ROUTH (C. H. F.) <i>remarks</i>	38, 57, 224
SANSOM (A. E.) the irregular heart: a clinical study	100
—— <i>remarks</i>	71, 115
SAVAGE (G. H.) symptoms of mental dissolution	252
—— <i>remarks</i>	263, 271
SHEILD (A. Marmaduke) on hæmorrhage from ulcerating	
bubo of the groin	318
—— case of cystic tumour of the auricle	348
—— ——— ulceration of the edges of the ears	349
—— ——— for diagnosis	353
—— ——— of congenital tumour in an infant	360
—— ——— warty growths	360
—— <i>remarks</i>	18, 30, 62, 237, 323
SIMPSON (W. J.) <i>remarks</i>	96
Skull, case of fracture of (H. W. Allingham)	351
SMITH (Heywood) <i>remarks</i>	57
SMITH (Solomon) <i>remarks</i>	79, 115, 244, 271
SMITH (T. Gilbert) on the diagnostic significance of hæmo-	
ptysis in aortic aneurysm	324
Speech, extreme defect of (W. B. Hadden)	350
Spleen, case of chronic enlargement of the (E. Cautley)	358
SQUIRE (Balmanno) <i>remarks</i>	242
STARTIN (Jas.) case of acne-keloid	352
STAVELEY (W. H. C.) <i>remarks</i>	19
STONHAM (C.) case of keloid	351
—— ——— congenital sacral tumour	351
—— ——— arterio-venous aneurysm	367
SUTTON (J. Bland) tubal moles and tubal abortions	48
—— <i>remarks</i>	58
SYMONDS (H. P.) case after amputation of the hip-joint	366
Syphilide, probable, refractory to treatment, case of (P. S.	
Abraham)	358

	PAGE
Syphilis, congenital, as a cause of nervous diseases in children (W. B. Hadden) . . . . .	59
Syphilitic affections of the nervous system (Lettsomian Lectures, by J. S. Bristowe) . . . . .	116
THOMPSON (E. Symes) <i>remarks</i> . . . . .	20, 30, 224
THOROWGOOD (J. C.) <i>remarks</i> . . . . .	114
Tumour, two cases of abdominal section for (W. H. Battle) .	244
—— bony, of the ascending ramus of the ischium (G. R. Turner). . . . .	346
—— pulsatile, of the neck (G. A. Herschell) . . . . .	348
—— cystic, of the auricle (A. M. Sheild) . . . . .	348
—— sacral, congenital (C. Stonham) . . . . .	351
—— congenital, in an infant (A. M. Sheild) . . . . .	360
TURNER (George R.) a case of volvulus of the small intestine	16
—— a case of bony tumour of the ascending ramus of the ischium . . . . .	346
—— <i>remarks</i> . . . . .	20
Ulcer, rodent, case of plastic operation for (J. Hutchinson, Jun.) . . . . .	367
Urethra, case of traumatic rupture of the (A. P. Gould) .	357
Volvulus of the small intestine, case of (G. R. Turner) .	16
Vulvo-vaginal glands, surgical treatment of cysts of the (A. Doran) . . . . .	38
WARNER (Francis) constitutional differences between boys and girls, and their relation to educational requirements	263
—— <i>remarks</i> . . . . .	262, 271
WATSON (W. Spencer) case of nasal polypus . . . . .	363
—— <i>remarks</i> . . . . .	277
WHEATON (S. W.) <i>remarks</i> . . . . .	63
WHITE (Hale) <i>remarks</i> . . . . .	212, 350
WOAKES (E.) <i>remarks</i> . . . . .	114

